TRANSACTIONS

OF THE

TWENTY-NINTH ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

HELD AT

MONTREAL, CANADA,

SEPTEMBER 16-20, 1924

The Thirtieth Annual Meeting of the American Academy of Ophthalmology and Oto-Laryngology will be held at Chicago, Ill., October 12-17, 1925. The attention of the members is called to the ruling of the Council, that when a paper published in the Transactions is illustrated, the author must pay the total cost of all colored plates, and one-half of the cost of all other cuts. The Editor will furnish an estimate of the cost of such cuts and plates upon request.

Only those members will receive the Transactions who have paid their dues by March 1st of the year for which the Transactions are published.

Printing arrangements make this rule of the Academy necessary.

Members are requested to notify the secretary of any error or omission in names, addresses or specialties in the list of members; also, of any change in address before the time of the 1925 meeting.

Please fill out slip enclosed with Transactions.

Printed and bound by
TUCKER-KENWORTHY COMPANY
Chicago, Ill.

Amer. Acad. of Ophthalmology
4-25-1925

OFFICERS FOR 1925

DR. HORACE NEWHART .....President
Minneapolis, Minn.

DR. EDWARD C. ELLETT.....President-Elect
Memphis, Tenn.

DR. JOHN M. WHEELER.....First Vice-President
New York City, N. Y.

DR. FRANK BRAWLEY.....Second Vice-President
Chicago, Ill.

DR. WILLIAM H. ROBERTS.....Third Vice-President
Pasadena, Calif.

DR. SECORD H. LARGE.....Treasurer
Cleveland, Ohio

DR. LUTHER C. PETER.....Secretary
Philadelphia, Pa.

DR. CLARENCE LOEB.....Editor of Transactions
Chicago, Ill.

MEMBERS OF COUNCIL

(EX-OFFICIO)

DR. HORACE NEWHART.....Minneapolis, Minn.

DR. EDWARD C. ELLETT.....Memphis, Tenn.

DR. JOHN M. WHEELER.....New York, N. Y.

DR. SECORD H. LARGE.....Cleveland, Ohio

DR. WALTER R. PARKER.....Detroit, Mich.

DR. THOMAS E. CARMODY.....Denver, Colo.

DR. WALTER B. LANCASTER.....Boston, Mass.

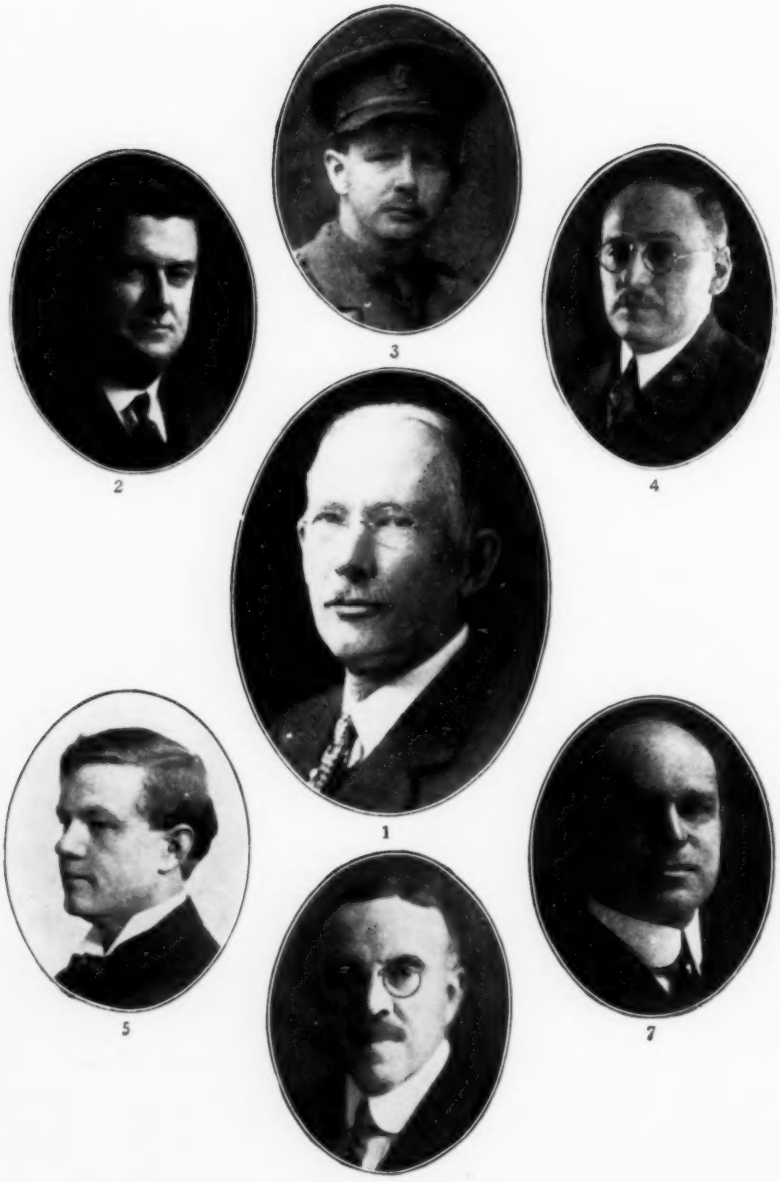
DR. FRANK L. DENNIS.....Colorado Springs, Colo.

DR. ARTHUR J. BEDELL.....Albany, N. Y.

DR. S. HANFORD MCKEE.....Montreal, Can.

OFFICERS FOR 1925.

1. DR. HORACE NEWHART, President, Minneapolis, Minn.
2. DR. EDWARD C. ELLETT, President-Elect, Memphis, Tenn.
3. DR. JOHN M. WHEELER, First Vice-President, New York City, N. Y.
4. DR. FRANK BRAWLEY, Second Vice-President, Chicago, Ill.
5. DR. WILLIAM H. ROBERTS, Third Vice-President, Pasadena, Calif.
6. DR. SECORD H. LARGE, Treasurer, Cleveland, Ohio.
7. DR. LUTHER C. PETER, Secretary, Philadelphia, Pa.
8. DR. CLARENCE LOEB, Editor of Transactions, Chicago, Ill.



OFFICERS FOR 1924.

1. DR. WALTER B. LANCASTER, President, Boston, Mass.
2. DR. WILLIAM P. WHERRY, First Vice-president, Omaha, Neb.
3. DR. S. HANFORD McKEE, Second Vice-president, Montreal, Can.
4. DR. HARRY L. POLLOCK, Third Vice-President, Chicago, Ill.
5. DR. SECORD H. LARGE, Treasurer, Cleveland, Ohio.
6. DR. LUTHER C. PETER, Secretary, Philadelphia, Pa.
7. DR. CLARENCE LOEB, Editor of Transactions, Chicago, Ill.

OFFICERS FOR 1924

Dr. Walter B. Lancaster.....President
Boston, Mass.

Dr. William P. Wherry.....First Vice-President
Omaha, Neb.

Dr. S. Hanford McKee.....Second Vice-President
Montreal, Can.

Dr. Harry L. Pollock.....Third Vice-President
Chicago, Ill.

Dr. Secord H. Large.....Treasurer
Cleveland, Ohio

Dr. Luther C. Peter.....Secretary
Philadelphia, Pa.

Dr. Clarence Loeb.....Editor of Transactions
Chicago, Ill.

MEMBERS OF COUNCIL

(EX-OFFICIO)

Dr. Walter B. Lancaster.....Boston, Mass.

Dr. William P. Wherry.....Omaha, Neb.

Dr. Secord H. Large.....Cleveland, Ohio

Dr. Walter R. Parker.....Detroit, Mich.

Dr. Thomas E. Carmody.....Denver, Colo.

Dr. Joseph L. McCool.....Portland, Ore.

Dr. William R. Murray.....Minneapolis, Minn.

Dr. Frank L. Dennis.....Colorado Springs, Colo.

Dr. Arthur J. Bedell.....Albany, N. Y.

PAST OFFICERS OF THE ACADEMY

Date	President	First Vice-President	Treasurer	Secretary	Meeting Place
1896..	Dr. Adolph Alt St. Louis, Mo.	Dr. W. C. Pipino Des Moines, Ia.	Dr. W. L. Dayton Lincoln, Neb.	Dr. Hal Foster Kansas City, Mo.	Kansas City
1897..	Dr. Adolph Alt St. Louis, Mo.	Dr. W. C. Pipino Des Moines, Ia.	Dr. W. L. Dayton Lincoln, Neb.	Dr. Hal Foster Kansas City, Mo.	St. Louis
1898..	Dr. B. E. Fryer Kansas City, Mo.	Dr. J. E. Colburn Chicago, Ill.	Dr. W. L. Dayton Lincoln, Neb.	Dr. Hal Foster Kansas City, Mo.	Chicago
1899..	Dr. J. E. Colburn Chicago, Ill.	Dr. W. Scheppegrell St. Louis, Mo.	Dr. W. L. Dayton Lincoln, Neb.	Dr. Thos. A. Woodruff Chicago, Ill.	New Orleans
1900..	Dr. W. Scheppegrell St. Louis, Mo.	Dr. M. A. Goldstein St. Louis, Mo.	Dr. W. L. Dayton Lincoln, Neb.	Dr. Fayette C. Ewing St. Louis, Mo.	St. Louis
1901..	Dr. M. A. Goldstein St. Louis, Mo.	Dr. H. V. Würdemann Milwaukee, Wis.	Dr. W. L. Dayton Lincoln, Neb.	Dr. W. L. Ballenger Chicago, Ill.	Cincinnati
1902..	Dr. C. R. Holmes Cincinnati, Ohio	Dr. W. L. Dayton Lincoln, Neb.	Dr. Otto Stein Chicago, Ill.	Dr. W. L. Ballenger Chicago, Ill.	Chicago
1903..	Dr. W. L. Ballenger Chicago, Ill.	Dr. J. C. Stilson Indianapolis, Ind.	Dr. Otto Stein Chicago, Ill.	Dr. Derrick Vail Cincinnati, Ohio	Indianapolis
1904..	Dr. Edward Jackson Denver, Colo.	Dr. Dudley Reynolds Louisville, Ky.	Dr. Otto Stein Chicago, Ill.	Dr. Derrick Vail Cincinnati, Ohio	Denver
1905..	Dr. Hanau W. Loeb St. Louis, Mo.	Dr. Derrick Vail Cincinnati, Ohio	Dr. Otto Stein Chicago, Ill.	Dr. Geo. F. Suker Chicago, Ill.	Buffalo
1906..	Dr. Casey A. Wood Chicago, Ill.	Dr. J. A. Stucky Lexington, Ky.	Dr. Otto Stein Chicago, Ill.	Dr. Geo. F. Suker Chicago, Ill.	St. Clair
1907..	Dr. J. A. Stucky Lexington, Ky.	Dr. R. A. Reeve Toronto, Canada	Dr. Otto Stein Chicago, Ill.	Dr. Geo. F. Suker Chicago, Ill.	Louisville
1908—	Dr. Derrick T. Vail Cincinnati, Ohio	Dr. Joseph Beck Chicago, Ill.	Dr. Otto Stein Chicago, Ill.	Dr. Geo. F. Suker Chicago, Ill.	Cleveland
1909..	Dr. Otto Stein Chicago, Ill.	Dr. Percy Fridenberg New York City	Dr. S. H. Large Cleveland, Ohio	Dr. Geo. F. Suker Chicago, Ill.	New York City

1910.. Dr. Wendell Reber Philadelphia, Pa.	Dr. L. B. Lockhard Denver, Colo.	Dr. S. H. Large Cleveland, Ohio	Dr. Geo. F. Suker Chicago, Ill.	Cincinnati
1911.. Dr. J. J. Kyle Indianapolis, Ind.	Dr. F. Park Lewis Buffalo, N. Y.	Dr. S. H. Large Cleveland, Ohio	Dr. Geo. F. Suker Chicago, Ill.	Indianapolis
1912.. Dr. Geo. F. Suker Chicago, Ill.	Dr. J. W. Murphy Cincinnati, Ohio	Dr. S. H. Large Cleveland, Ohio	Dr. L. M. Francis Buffalo, N. Y.	Niagara Falls, Ont.
1913.. Dr. J. W. Murphy Cincinnati, Ohio	Dr. J. O. McReynolds Dallas, Texas	Dr. S. H. Large Cleveland, Ohio	Dr. L. M. Francis Buffalo, N. Y.	Chattanooga
1914.. Dr. J. M. Ray Louisville, Ky.	Dr. J. M. Ingersoll Cleveland, Ohio	Dr. S. H. Large Cleveland, Ohio	Dr. L. M. Francis Buffalo, N. Y.	Boston
1915.. Dr. Joseph Beck Chicago, Ill.	Dr. Robert S. Lamb Washington, D. C.	Dr. S. H. Large Cleveland, Ohio	Dr. L. M. Francis Buffalo, N. Y.	Chicago
1916.. Dr. J. E. Brown Columbus, Ohio	Dr. T. E. Carmody Denver, Colo.	Dr. S. H. Large Cleveland, Ohio	Dr. L. M. Francis Buffalo, N. Y.	Memphis
1917.. Dr. W. L. Dayton Lincoln, Neb.	Dr. Edw. Stieren Pittsburgn, Pa.	Dr. S. H. Large Cleveland, Ohio	Dr. L. M. Francis Buffalo, N. Y.	Pittsburgh
1918.. Lt. Col. Allen Greenwood Boston, Mass.	Dr. Virginius Dabney Washington. D. C.	Dr. S. H. Large Cleveland, Ohio	Dr. L. M. Francis Buffalo, N. Y.	Denver
1919.. Dr. J. M. Ingersoll Cleveland, Ohio	Dr. Melville Black Denver, Colo.	Dr. S. H. Large Cleveland, Ohio	Dr. Luther C. Peter Philadelphia, Pa.	Cleveland
1920.. Dr. L. M.Francis Buffalo, N. Y.	Dr. Hal Foster Kansas City, Mo.	Dr. S. H. Large Cleveland, Ohio	Dr. Luther C. Peter Philadelphia, Pa.	Kansas City
1921.. Dr. Emil Mayer New York City	Dr. John R. Newcomb Indianapolis, Ind.	Dr. S. H. Large Cleveland, Ohio	Dr. Luther C. Peter Philadelphia, Pa.	Philadelphia
1922.. Dr. Walter R. Parker Detroit, Mich.	Dr. Ross H. Skillern Philadelphia, Pa.	Dr. S. H. Large Cleveland, Ohio	Dr. Luther C. Peter Philadelphia, Pa.	Minneapolis and St. Paul
1923.. Dr. Thomas E. Carmody Denver, Colo.	Dr. Hunter H. McGuire Winchester, Va.	Dr. S. H. Large Cleveland, Ohio	Dr. Luther C. Peter Philadelphia, Pa.	Washington, D. C.
1924.. Dr. Walter B. Lancaster Boston, Mass.	Dr. William P. Wherry Omaha, Neb.	Dr. S. H. Large Cleveland, Ohio	Dr. Luther C. Peter Philadelphia, Pa.	Montreal, Canada

JOINT SESSIONS

TABLE OF CONTENTS

PAGE

Address of Welcome.
SIR ARTHUR W. CURRIE, G.C.M.G., K.C.B.
Montreal, Canada..... 1

President's Address.
WALTER B. LANCASTER, M.D., Boston, Mass..... 4

Minor and Major Neuralgias of the Fifth Nerve.
JOHN F. BARNHILL, M.D., Indianapolis, Ind..... 9

Nonrefractive Asthenopia.
WILLIAM THORNWALL DAVIS, M.D.,
Washington, D. C..... 40

Some Aspects of Primary Cataract.
LT. COL. HENRY KIRKPATRICK, London, Eng..... 61

The Mechanism of Papilledema.
WALTER R. PARKER, M.D., Detroit, Mich..... 77

The Significance of Papilledema to the Ophthalmologist.
JAMES BORDLEY, JR., M.D., Baltimore, Md..... 82

Papilledema in Oto-Laryngology.
LEON E. WHITE, M.D., Boston, Mass..... 89

The Significance of Papilledema to the Neurologic Surgeon.
GILBERT HORRAX, M.D., Boston, Mass..... 103

OPHTHALMOLOGIC DIVISION

TABLE OF CONTENTS

PAGE

Nonoperative Treatment of Cataract.
ALLEN GREENWOOD, M.D., Boston, Mass..... 125

Bacteriologic Examination of the Conjunctival Sac as a Routine Measure in the Preoperative Technic.
W. G. M. BYERS, M.D., and A. A. BRUÉRE, M.D., Montreal, Canada..... 141

Secondary Cataract Opening by Single Straight Incision.
JOHN M. WHEELER, M.D., New York City, N. Y... 149

The Value of the Fusion Faculty and Methods for Its Development.
DAVID W. WELLS, M.D., Boston, Mass..... 159

Congenital Abduction Deficiency.
HAROLD GIFFORD, M.D., Omaha, Neb..... 176

The Etiology of Phlyctenular Keratoconjunctivitis.
W. W. BLAIR, M.D., Pittsburgh, Pa..... 195

Etiology and Treatment of Scleritis and Episcleritis.
WILLIAM L. BENEDICT, M.D., Rochester, Minn... 211

Sensitive Test Charts for Refraction.
JONAS. S. FRIEDENWALD, M.D., Baltimore, Md..... 222

OTO-LARYNGOLOGIC DIVISION

TABLE OF CONTENTS

PAGE

Medical Remedies for the Control of Hemorrhage.
SAMUEL G. HIGGINS, M.D., Milwaukee, Wis..... 231

The Treatment of Deformities of the Nose Due to Accidental and Operative Traumatism.
WILLIAM WESLEY CARTER, M.D.,
New York City, N. Y..... 244

The Gradenigo Syndrome, With Report of Three Cases.
WILLIAM HARDIN SEARS, M.D., Huntingdon, Pa... 264

The Treatment of Paranasal Sinus Disease in Children.
MILLARD F. ARBUCKLE, M.D., St. Louis, Mo..... 283

Bell's Palsy: Four Cases of Infectious Origin.
VIRGINIUS DABNEY, M.D., Washington, D. C..... 294

A Further Study of the Toxic Effects of Local Anesthesia.
EMIL MAYER, M.D., New York City, N.Y..... 303

Bronchopneumonia and Abscess Secondary to Tonsillectomy.
JUDSON DALAND, M.D., Philadelphia, Pa..... 319

Ciliary Inhibition or Destruction in Tracheobronchial Asthma, With Notes on Bronchoscopic Treatment.
WILLIAM F. MOORE, M.D., Philadelphia, Pa..... 333

Observations on Fifty Cases of Foreign Bodies in the Air and Food Passages.
GABRIEL TUCKER, M.D., Philadelphia, Pa..... 348

The Present Status of Electrotherapeutic Measures Used in the Practice of Oto-Laryngology.
J. C. Beck, M.D., and H. L. Pollock, M.D., Chicago, Ill..... 400

Tonsillectomy and Adenoidectomy in Selected Cases During the Periods of Primary and Secondary Infections.
Frederick T. Clark, M.D., Westfield, Mass.... 413

New Instruments and Appliances..... 429

Motor Driven Antrum Drill.
Roy S. Blinkley, M.D., Dayton, Ohio..... 429

Handle and Canulae for Suction and Irrigation.
J. C. Beck, M.D., H. L. Pollock, M.D., and F. L. Lederer, M.D., Chicago, Ill..... 430

Cautery Handle and Tips.
J. C. Beck, M.D., H. L. Pollock, M.D., and F. L. Lederer, M.D., Chicago, Ill..... 432

The Visibility of the Retinal Capillaries in Ophthalmoscopy with Yellow-Green Light.
Jonas S. Friedenwald, M.D., Baltimore, Md.... 434

Necrology 440

Minutes 441

Constitution 473

By-Laws 475

Alphabetic List of Members..... 481

Geographic List of Members..... 517

Index of Authors and Subjects..... 561

TRANSACTIONS

OF THE

TWENTY-NINTH ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

JOINT SESSIONS

ADDRESS OF WELCOME.

SIR ARTHUR W. CURRIE, G.C.M.G., K.C.B., PRINCIPAL AND VICE CHANCELLOR, MCGILL UNIVERSITY.

MONTREAL, CANADA.

Mr. Chairman, Ladies and Gentlemen: I deeply appreciate the honor and privilege, as Principal of McGill University, of saying a few words and extending a sincere and gladsome welcome to the members of this society, their distinguished guests and visitors. Doubtless you are all well aware, that the holding of conferences and conventions is no longer a novelty in this metropolis of Canada. Particularly in recent years has Montreal become a very Mecca for conventions. I have an old friend who is the doorkeeper at one of the leading hotels here, who is something of a philosopher. He is of the opinion that the holding of these conventions in our city is rather spoiling Montreal. When I asked him the reason why, he said the best people no longer come here; and when I endeavored to find out what he meant by the "best people," he said the best people are those who drive up in their own private cars, with their own chauffeurs. I do not know whether, if that classification were to stand, you would be regarded as the "best people" or not, but despite what the doorkeeper says about conventions, I want to assure you that no society that has ever met in Montreal has received a more sincere and respectful welcome, and that is quite proper, because you come not for the advancement of yourselves, but rather that you may do something to help humanity. You have come to report and to confer upon the best measures and methods of lessening human suffering and alleviating human pain. You have not come, as some others have, because we are a city rather far removed from the arid belt, but rather, I take it, because from the early days of Canadian education, Montreal has made earnest efforts to make some contribution to the advancement of medical science. With those earnest efforts there have been associated men whose names stand high on the world's roll of honor, men whose contributions will be of lasting benefit to humanity, men whose lives

marked a veritable epoch in the profession to which you belong.

I am glad to say that the first degree granted by McGill University was granted to a graduate in medicine, and that the graduates in that profession are larger by far than the graduates of any other department in the University.

Now, since those early days, more than one hundred years ago, days of scant support, of inadequate facilities and primitive methods, McGill has gone steadily forward in research and in endeavor to do something that may be regarded as a positive contribution to medical knowledge. You will visit our University and Medical School, and there you will find tablets testifying to that fact. You will meet many men, men who are daily witnesses to the students of today of the strength of their devotion to their ideals. And so you meet here surrounded by traditions of which we in Canada are proud.

It may interest you to know that investment of our Medical School, its upkeep and operation, represents an outlay of quite ten millions. In the last four years, we have added considerably to our equipment. You will visit the principal medical building, the gift of Lord Strathcona, one of the greatest benefactors of the University, and there you will find housed the Department of Anatomy, presided over by Prof. S. E. Whitnall, and the Department of Histology, presided over by Professor Simpson. And I would like you to look at the room we have dedicated to Sir William Osler. Close by, I hope you will visit our Biological Building, and the other various buildings. We have this year erected, and will open in a few weeks, our new Pathological Institute, which we believe is one of the finest of its kind in existence. Our Department of Pathology, presided over by Dr. Oertel, is very closely associated with the Royal Victoria Hospital, one of the best in the world, and it is one evidence of our close cooperation with that institution that we have just established a clinic there which will be presided over by Professor Jonathan Meakins, formerly of the University of Edinburgh. I am glad to say we have also very close association with another large hospital here, the Montreal General Hospital. There is coming to that hospital, as head of the Department of Medicine, Professor Palmer Howard, a man whom we welcome back to Montreal with great heartiness, and look forward to a career of great

usefulness for him! We have also close association with the other hospitals in the city.

I shall not detain you from your deliberations. I am an old soldier, and I have a peculiar sympathy with you in your fight against disease. I congratulate you on the wonderful victories already won and the glorious results achieved. Some of the deadliest enemies of human health you have entirely overcome; others are under control, and others have reached the vanishing point. Such splendid results could not have been attained except by the unceasing toil and earnest endeavor, without clash of cymbals or blare of trumpets, which you devote to your task, with only the reward of conscientious service.

I will close with the immortal Stevenson's tribute to the physician.

"He is the flower of our civilization; and when that stage of man is done with, and only remembered to be marveled at in history, he will be thought to have shared as little as any in the defects of the period, and most notably exhibited the virtues of the race. Generosity he has, such as is possible to those who practice an art, never to those who drive a trade, discretion, tested by a hundred secrets; tact, tried in a thousand embarrassments; and what are more important, Herculean cheerfulness and courage."

May this conference of yours be wonderfully useful, and may God bless you all.

PRESIDENT'S ADDRESS.

WALTER B. LANCASTER, M.D.

BOSTON, MASSACHUSETTS.

Fellows of the Academy, Honored Guest and Distinguished Visitors, Ladies and Gentlemen:—Welcome to the 29th meeting of the American Academy of Ophthalmology and Oto-Laryngology!

To our colleagues of Canada, the City and the Empire they represent, our warmest greetings and our heartiest thanks for their invitation to meet in this beautiful environment, and our sincerest appreciation of their wonderful hospitality. May this meeting weld one more link in the chain that binds our two countries in a friendship no storms can shatter, no misunderstandings undermine—a friendship which, like all true and great friendships, is rooted in mutual service, mutual respect, mutual advantage and profit.

A special word of welcome to those who are for the first time present at a meeting of this Academy, whether as new members or as visitors. It is our wish that you feel the genuineness of our good fellowship, the breadth of our democratic spirit, and above all, that you partake of the inspiration and enthusiasm that I feel sure will characterize these meetings.

Let me sound the keynote of our meetings—rather let me sound the major chord on which the harmony will be founded. The first note of the chord is Enthusiasm! Note the derivation of the word—ἐν θεος—a god within. As Pasteur exclaimed, after thanking the Greeks for one of the most beautiful words in our language, *"Heureux celui qui porte en soi un dieu."* Enthusiasm is the divine fire. It burns in every soul, but to attain its highest intensity, it needs the contact of many. Compare the heat from one lump of coal with that produced by many in contact.

Enthusiasm is contagious, therefore let us expose ourselves freely to the virus, let us tolerate no quarantine, let us overcome even our natural repugnance to exuberant feelings, that by some means, by all means, we may fan the flame

of that divine fire, that we may be found to harbor the god within. *"Heureux celui qui porte en soi un dieu."*

Enthusiasm is the urge toward *service*, the greatest of our aims as physicians. It is the urge toward *research*, the greatest of the benefits we can confer upon our world. No ship can reach port, no matter how wisely navigated, unless it has motive power, either wind without or engine within. Enthusiasm is the motive power essential to any progress, any attainment. It is enthusiasm that makes possible the steady grind of monotonous work when results are slow. It is enthusiasm that underlies that infinite capacity for taking pains which spells genius.

I would not belittle the part of the wise navigator who steers the ship; therefore, I take for my second note in the chord, Truth. Let it be our constant aim to seek for the truth, for more wisdom, more knowledge. Let us strive to sift out and reject error. Let the Academy stand for the highest grade of scholarship, with no tolerance for shams, though we have only sympathy for error and an earnest desire to help ignorance, which we all humbly admit we share. Let there be no snobbish superiority of greater wisdom when all are so far short of perfection. Effective knowledge may be expressed as a fraction. It is equal to the number of true facts divided by the number of untrue facts. Each "fact" that is not true distorts all the facts that are true. The Academy will ever strive to increase the true facts and to scotch and bury the untrue facts.

There are many beautiful notes clamoring for the third note of the chord, but I must not prolong this into an overture. Let me take Thoroughness for the third note. Let me urge that it be our daily practice. How many of our mistakes in diagnosis must be laid to lack of thoroughness! How many of our operative complications! How many of our imperfect therapeutic results! Who among us has not had the humiliation of having a case of ours sent back from a consultant with the correct diagnosis, which we could have made just as well ourselves if we had only been thorough? It is doubly humiliating, for we not only fail in our diagnosis as ophthalmologists, but we fail in our duty to our patient as a physician in whom the patient trusted, to whom he committed his eyes, believing we would do our best. When work becomes too pressing, or our strength inadequate for thorough, that is,

our best work, it is our duty to restrict our practice to what can be done conscientiously and thoroughly. When the cause of lack of thoroughness is lack of enthusiasm, then attend more meetings of the Academy, or read the Transactions.

The last note in the chord, the dominant note, the note with the wonderful harmonics which impart those elusive characteristics which make up perfection of tone quality—the last note is Honor. The Academy stands ever more and more inflexibly for high standards of honor—professional honor and, fundamental to all, personal honor! Those qualities which mark the gentleman, and which, I believe, reach their highest flower in the English and French speaking peoples! Let us live up to the high honor of the vocation to which we are called. Let us strive to be worthy of those great exemplars of our profession, that each of us may humbly say: I, too, am a physician and a gentleman.

It is customary for the President to say a few words about the arrangements. I would rather let them speak for themselves. Our meeting place, our halls and rooms, our program, all bespeak the efficient, untiring work of your officers. If I may single out two or three, they are Doctor McKee, the chairman of the local committee, whose work has been prodigious and whose accomplishment incredible; the Secretary, whose labors are appreciated only by those inside, and the First Vice-President, a giant himself in body and mind, whose name should not be "wherry", which according to the dictionary is "a small boat propelled by hand", useful as that little boat is, but rather it should suggest a submarine whose work is often out of sight, but whose results are tremendous.

It is the President's duty to make recommendations for the good of the Academy and of the profession in general, He has this on his mind in a general way during his year of office. One recommendation is being carried out—the voice amplification.

Second, the incoming President is often, as was the case with myself, ignorant of his duties to a degree which hampers his usefulness. Why not let the outgoing President hold over to cooperate with the President, who will then take full charge only after a period of guidance and helpful assistance by his predecessor?

Third, is there any suggestion I can make for the welfare of ophthalmology in general and this Academy in particular?

In attempting to do so I hark back to the days when I joined the Academy eleven years ago. The American Board for Ophthalmic Examinations was in the borning. We were all eager to see better teaching of our specialty. Indeed, it was to elevate the standards of teaching ophthalmology more than from any other motive, that the first steps were taken a dozen years ago which led to the organization of this Board destined soon to be followed by a somewhat similar Board in Oto-Laryngology. I was one of those who were disappointed that it took so long for this movement to be felt in the University medical schools, but the forward urge is unmistakable today.

Now I am led to take another survey of the field, to consider the training in ophthalmology today (not being competent to include oto-laryngology) and see if there is some weak point that we may perhaps strengthen to the lasting benefit of American ophthalmology and to the glory of this Academy.

I have no hesitation in saying that most departments of Ophthalmology are very well taught today in many places in this country. To Denver belongs the honor of leadership under Jackson, for where Jackson sits there is the head of the table. To Philadelphia, with its peerless faculty, belongs the honor of the highest attainment thus far reached. We have only to compare the present with conditions a dozen years ago, to see what splendid progress has been made.

Now where is there a weak point which we can perhaps strengthen? I think there is no question that the weakest point is the physiology of vision. This seems to me so obvious as to need no elaboration. Why is this department so weak? The answer is not because it is unimportant, for it is absolutely fundamental and indispensable; not because there is not much known about it, for the field has been and is being worked by some of the most brilliant, profound and productive minds that have touched the science of ophthalmology in any of its branches. No, it is from no lack of material, no lack of importance. It is simply and solely from lack of competent available teachers. Several schools would eagerly seize the opportunity to include adequate courses in the various departments of physiology were teachers available. Is it not clear that we need well trained, competent ophthalmic teachers of the physiology of vision? How can we get them?

I have a proposal for your consideration. We have a fund available for the advancement of ophthalmology and oto-laryngology. Speaking now for the former, what an opportunity to take in hand a young man of promise, who has had suitable preliminary training, and grant him a fellowship for three or four years for the study of this branch of ophthalmology. Let him spend a few months here, a few months there, wherever opportunity exists for some subdivision of his subject, whether under psychologist, physicist, biologist, or pathologist. During this time clinical ophthalmology should not be entirely neglected, for our ideal teacher must know the clinical as well as the physiologic and the laboratory side. After such a period, he would be prepared to found a department of the physiology of vision and would be an eagerly sought man. He would be assured at once of prominence in his field, high standing in all societies, and great opportunity to raise the standards of ophthalmology by training teachers, by being the nucleus of a growing group of workers—an American School of the Physiology of Vision, whose foundations were laid by this Academy.

In conclusion, let me assure you of my deep appreciation of the distinguished honor you have conferred upon me in making me your President, and charging me with the responsibilities of that important office, which I am so imperfectly fitted to perform. I can say only that I have the interests of the Academy at heart, and shall ever strive to advance the Academy toward the great future which I believe is its destiny.

MINOR AND MAJOR NEURALGIAS OF THE HEAD.

JOHN F. BARNHILL, M. D., F. A. C. S.

INDIANAPOLIS, IND.

The fifth nerve supplies sensation to all of the most important structures of the head. Pain, the symptom which most frequently brings the patient to the surgeon, is a disease of this nerve, or of the tissues which it supplies. The importance, therefore, of a study of the question of trifacial neuralgia is exceeded by no other subject related to diseases occurring in the wide areas of distribution of this nerve. In the pursuit of such study, one is constantly reminded of the fact, that the neuralgias of the head are more or less closely associated with disturbances of the sympathetic nerve supply to the same regions, and that, because of a present lack of knowledge concerning this system, the exact influence cannot be analyzed or properly interpreted.

Disease of the fifth nerve, trigeminal neuralgia, epileptiform neuralgia, "trigger" neuralgia, tic douloureux, names commonly used to designate different diseases, at least varying degrees or type of the same disease, is of frequent occurrence. A correct classification of the different types of the affection is essential for purposes of study, and, what is of greater importance, for the determination of a suitable plan of treatment for each. Such classification may at present, perhaps, be most sensibly based on the symptoms, and the two varieties most acceptable are the major and minor neuralgias, since all cases fall rather clearly into one or the other class. Those that should not be so grouped are most commonly of a variety more or less troublesome to make out, cases that merge so closely that definition is lost; or of that somewhat rarer class which is often referred to as hysteroid type.

The symptoms of minor neuralgia are less paroxysmal and less terrifying than those of the major variety. The pain may be of a dull aching character, with occasional lightning like shifts from some center. It is in nearly every instance continuous. It may be but a nerve "soreness," a nerve "ache," in

which case there likely may be found points of tenderness to pressure over the nerve exits. In no instance of minor neuralgia will there be a "strike" in some parts of the head accompanied by a "tic" or spasm of the part. The pain may occasionally affect both sides. In most instances a cause may be found by painstaking examination. This cause may be a decaying or abscessed tooth. It may be due to a suppuration in any of the paranasal sinuses. It may be a symptom of herpes zoster, or it may arise from eyestrain, glaucoma, or other ocular disease. In short, peripheral nerve irritation or nerve infection may be present. Again, some general affection, as anemia, malaria, functional disturbances of menstruation or other bodily malady may clearly be present as a cause, at least a predisposing cause. The one point which is usually demonstrable in minor neuralgia, is that persistent effort by means of physical examination and laboratory tests will enable the surgeons to determine some actual focus of disease, some satisfactory reason for the neuralgic pain.

In major neuralgia, the pain is always, even in the worst cases, more or less intermittent. There are, in most instances, two periods of intermission. One, the long period, is the interval between the attacks, the period when the patient is entirely free from pain and is apparently normal in health. In the beginning of the major type, these periods of freedom are often of long duration, sometimes weeks or even months. However, as the disease progresses, the intervals shorten, until in the worst types this period is entirely wanting. The short period of intermission is the one that occurs during the "attacks." The long period having ended after a freedom lasting days, weeks or even months, an "attack" comes on. This attack is, however, not a continuous pain, but rather a series of "strikes," "tics" or epileptiform seizures of pain, which lasts for a few seconds, occasionally as long as a minute, and is followed by the short interval of complete freedom from all suffering. This latter interval varies in duration from a few minutes, or even less in extreme cases, to a few hours. The patient, while enjoying this interim of entire freedom from pain, is with extreme suddenness "struck" by a lightning like and terrifying pain in the affected region of the lower jaw, roof of the mouth, gums, superior maxilla, supraorbital region, or perhaps more than one of these areas in association. The area of such "strike" is definite, and the

patient is always able to define it accurately. Such pain is practically always on one side of the face and head. Adson states that 3% of cases are affected on both sides. The writer has seen but one such in about 300. Contrary to many who have described major neuralgia, neither the peripheral branches nor the superficial foramina of exit of the nerves upon the face and forehead are tender to pressure. On the contrary, deep pressure over the areas is often grateful to the sufferer. Strangely enough, the lightest possible touch of the affected surface often starts the painful attack. In men with beard or moustache, merely touching a hair, however lightly, may set off the pain. The touch of the skin with a feather, a breath of air upon the face or a sip of water in the mouth will do likewise, giving origin to the name "trigger neuralgia."

In searching for the cause of major neuralgia none is found. Teeth, sinuses, tonsils, nose and eyes are usually normal. If abnormal, the correction of the defect will have no effect upon the major neuralgia. This fact is amply proven by the many patients whose sound teeth have been extracted, sinuses curetted, or ethmoids "thoroughly exenterated," without checking in the slightest degree the advance of the neuralgia in what seems to be its normal progress from bad to worse.

Minor neuralgias may occur at any age, but are seen oftenest in those comparatively young. The major variety is met with most often in those past middle life. Among the writer's patients, the youngest was 32 and the oldest 84 years of age. Early writers gave the average age at about 50. Frazier makes the observation that undoubtedly major neuralgia now occurs more frequently and at an earlier age than formerly. If the fact can be established that no disease of any kind exists about the head of one suffering from neuralgia of a spasmodic type, this fact alone should be sufficient to classify the patient as a major and not a minor neuralgic. Major neuralgia is a disease without visible lesion, whereas minor neuralgias very frequently arise from discoverable causes.

Genuine neuralgia must be carefully distinguished from other kinds of head pain. There are borderline cases in which the diagnosis is not easy. A type related to hysteria is of this kind. The hysteroid cases of neuralgia which the writer has seen have had constant pain over some area of distribution

of one or more division of the fifth nerve. In none of these cases was there spasmodic pain. In none was there a "strike." In none was there a clearly defined local or general disease which was causative. In none did the alcoholic injection of the affected nerve or ganglion relieve the pain. In one, resection of the posterior root had no lasting effect.

Another type of head pain simulating major neuralgia is due to some intracranial disease other than of the fifth nerve or Gasserian ganglion. It may arise from a tumor or aneurism pressing on the Gasserian ganglion or its branches. Caminiti reports cases due to tumor of the pons, aneurism of the carotid and tumor of the brain. Kanavel has also reported several such instances, while Ernst Sachs reports an endothelioma attached to the Gasserian ganglion, in his own practice, and gives references to 37 similar cases that have been studied pathologically and classed as endothelioma, sarcoma or carcinoma. In 1921 Frazier stated that up to that time 43 such cases were on record, mostly of a malignant nature. Other surgeons have found osseous erosions of the skull in the vicinity of the Gasserian ganglion, or exostosis of the petrous portion of the temporal bone, so located that pressure on the ganglion was produced, with resulting pain. Pain in pseudoneuralgia types, especially from malignant tumors of the ganglion, is not spasmodic, does not "strike," there are no "trigger" areas over the distribution of the nerve, and the pain is not set up by trivial causes such as touch of a feather or a breath of air upon the "trigger zone." The pain may at first closely simulate a major neuralgia, but sooner or later trophic keratitis, hypesthesia and ocular palsies, together with other symptoms more or less common to tumors of the middle fossa of the brain, develop, and these rather clearly differentiate the disease from any form of neuralgia.

The pathology of tic douloureux is not constant. It has been a popular theory that the disease begins in the peripheral nerves and ascends, finally reaching the Gasserian ganglion. This theory seems not wholly borne out by facts. Spiller says, that whether the disease begins in the ganglion or in the peripheral nerves, is a question that can not be decided. Spiller and Caminiti report the collective examination of 27 Gasserian ganglia after their surgical removal, with the result that cellular changes in the ganglion are not constant, and when present are not the same in all cases. Beckman says

that 6 ganglia examined by Wilson showed marked inflammation in all. Frazier thinks it probable that the pathology of the minor neuralgias lies in the sphenopalatine ganglion, while that of the major neuralgias is confined solely to the Gasserian ganglion. Sharpe states that the pathology is obscure, and that in the majority of cases no lesion of either the Gasserian ganglion or its branches can be determined. Sharpe further believes, that in those cases which have been examined, in which degenerative changes have been found, the changes were often due to former treatment, especially to the injection of alcohol.

Assuming that Frazier is correct in the assumption that the pathology of minor neuralgia is centered in the sphenopalatine ganglion, whereas the Gasserian ganglion is the seat of the major variety, it is clear that the necessity for absolutely correct diagnosis of the particular type of neuralgia, in a given case, is entirely essential before treatment is undertaken.

Minor and pseudoneuralgias have been considered here for the purpose of a clear differentiation of each. Minor neuralgia may now be dismissed, with the statement that treatment for its relief and cure may wisely consist in the administration of drugs, and the employment of surgical measures directed to the teeth, tonsils, paranasal sinuses or sphenopalatine ganglion. None of these measures have any place in the treatment of tic douloureux. Frazier has no doubt stated the facts concerning the treatment of this latter disease, when he says that the treatment is now standardized: Alcohol injections for palliation, section of the sensory root for permanent cure. Surgeons of large experience at the present time admit the correctness of Frazier's view, with the sole exception of Hutchison, of England, who still advocates subtotal removal of the ganglion.

The treatment being limited to the injection of alcohol or to the severance of the posterior root of the ganglion, to what type of the disease is each method best applicable? Many opinions are available. Those most acceptable relate to the age of the patient, the severity and duration of the neuralgia, and the physical condition of the affected individual. In the early months of the disease, the patient is usually hopeful of recovery from the use of mild measures and hence chooses the alcohol injection. In case of those past 70, when

the patient can at best live but a few years, or if the physical condition of the sufferer is poor, the surgeon may wisely advocate the injection of the ganglion or its affected branches. However, in cases where there is a return of pain in a few weeks or months after such injection, or after repeated injection, especially in those in middle life or younger, the surgeon should advise section of the root, for the reason that such procedure is permanent in the relief of pain, and the risk to life is now exceedingly small.

Alcoholic injections of the peripheral ends of the nerves, or of the branches at their superficial foramina of exit, are no longer used. The present plan is to inject the alcohol into the nerve trunks at their exit from the foramen rotundum or the foramen ovale, or to inject it directly into the ganglion itself. No attempt is made to inject the first division of the ganglion as it emerges from the skull into the orbit, and the practice of injection of the branches of this division as they emerge upon the forehead at the supraorbital notch, is now practiced by but few.

The question arises, which is the better procedure, injection of the Gasserian ganglion, or injection of the nerve trunks at their foramina of exit from the skull? In case the third division of the nerve only is involved in the neuralgia, all agree that injection of this nerve at its exit is best. When two or more divisions are involved, there is sharp difference of opinion among those of large experience. A majority of surgeons believe that injection of the nerve trunks gives all the relief possible from the use of alcohol, while at the same time avoiding some of the dangers that arise from injecting the ganglion itself. That such dangers do arise from ganglionic injections of alcohol has been pointed out by many, including Beckman, Vaughn, Grinker, Härtel, Martin and Cushing. Such unfortunate results have been published as double vision due to oculomotor paralysis, stiffness of the jaw due to faulty injection or to paralysis of the motor root of the fifth, which supplies the muscles of mastication, and facial palsy due to complete paralysis of the facial nerve. Cushing reports 4 cases of complete facial palsy. The writer has seen two such cases; one injected by an associate, one case of his own. Keratitis sometimes follows the use of alcohol, when all three branches of the fifth are completely anesthetized. Loss of the eye from this cause has been re-

ported. Again, because of the close proximity of the Eustachian tube to the foramen ovale, the operator may pass the injection needle into this tube, and inject the alcohol into the tube and middle ear under the impression that the same is properly placed in the nerve trunk or ganglion. After such faulty injection, the ear has been seriously damaged, middle ear suppuration set up, and even labyrinthine involvement with great dizziness has followed. Three deaths from alcohol injections have been reported. Flesch states that injection of alcohol directly into the ganglion sets up eye troubles of some sort in 50% of cases, while Härtel believes that such affections do not occur from this cause in more than one fourth of those injected. Vaughn believes there is less danger to the eye from alcoholic injection of the ganglion than from sensory root resection, and quotes Wilder, who had 300 cases of injection of alcohol with only one case of keratitis.

The internal maxillary and the middle meningeal arteries, as well as the peterygoid plexus of veins, pass through or near the pathway of the needle from its external point of entrance to its deep destination in the ganglion. Any of these vessels may be transfixed or even entered by the needle, and, should injection of the alcohol be made into one of them, dangerous consequences likely will ensue. Of course, any danger from this source is quite as likely to follow the attempt to inject a deep nerve trunk as the ganglion itself. However, an added danger arises in ganglionic injections, should the needle be passed to too great a depth and at an incorrect angle, when the cavernous sinus may be entered and the alcohol be deposited into the blood stream at this point. Cushing has well said that alcohol injections for the cure of neuralgia "should be carried out with practical hands directed by the capacity to visualize deep anatomic structures," and adds further that "this is more or less a gift, and there are no rules to follow." That "there are no rules to follow" is not wholly true. Rules for making these injections may and have been formulated which are reasonably certain.

The writer recognizes the many complications and dangers encountered in making alcohol injections directly into the Gasserian ganglion. He, nevertheless, believes that this mode of treatment of major neuralgia is best, provided the method of alcohol injection is chosen. While it is probable that some of these dangers may not always be avoided, it is

believed that a definite knowledge of the anatomic structures on the part of the operator, and the acquirement of skill by practice, will obviate most of the serious objections that have been recorded, thus making of the procedure a certain means of prolonged relief at but slight risk. Skillfully carried out, injection of the ganglion will also obviate the necessity of root resection in many cases.

Methods of Injecting the Gasserian Ganglion

The patient should be in a hospital, for, while the injection

Fig. 1. Shows under surface of Gasserian ganglion with dural attachment and relationship of ganglion to motor root, to carotid artery and to third, fourth and sixth nerves. The dura propria has been incised and reflected from the surface of the ganglion.

itself is in no sense a major operation, the consequences of the same may amount to as much. Absolute sterility must be maintained, and this a hospital can best provide. One hour before the proposed injection of the alcohol, the patient is given a hypodermatic injection of H.M.C., the amount depending somewhat upon the weight and excitability of the patient, small persons receiving the tablet No. 2, while No. 1 is given if the individual is large. It is not desirable to render the patient wholly unconscious, but rather stuporous to the

point of being able to answer questions, yet wholly oblivious to pain except when severe. Hence, if at the time of making the injection the individual is yet fully conscious and sensitive to common pain, it is wise to give a second hypodermatic injection of one-half the previous dosage, and to wait 15 to 30 minutes. General anesthesia is never advisable. In view of the fact that an intracranial structure is to be penetrated by the injecting needle, it is of course essential to employ an absolutely sterile technic (Figs. 1 and 2). To this end,

Fig. 2. Shows dura mater normally attached in the anterior and middle fossa, with course of middle meningeal artery showing through. The dura in the posterior fossa has been elevated, incised and so reflected as to show the posterior root of the fifth nerve in its relationship to other important cranial nerves. It may be noted that the posterior root enters the fossa without any protecting dural sheath, and that, therefore, any fluid injected outside the nerve tissue itself would flow almost unhindered into the posterior fossa, where it would reach the third, fourth, sixth, seventh and eighth nerves, and paralyze their function.

the face, lower jaw and chin of the patient are treated with the ether, iodin and alcohol sequence. If the mouth, as here recommended, be chosen as the point of entrance of the needle, this cavity, including the teeth, if any, should be thoroughly scrubbed with an antiseptic, and the area of actual entrance of the needle should be painted with iodin. All other parts of the head are draped with sterile linen (Figs. 6 and 7). A half drachm of 2% solution of novocain should then be injected along the pathway to be followed by the needle used for carrying the alcohol. Novocain or other local anesthetic solution should not be injected into the

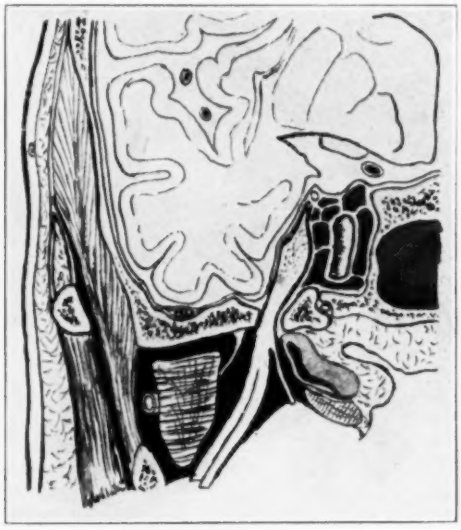


Fig. 3. Section of the head on a level with the Gasserian ganglion, showing the relationship to the cavernous sinus, brain and Eustachian tube.

nerve trunks or Gasserian ganglion previous to the alcohol injection. In several cases, the writer has had the experience of properly placing the needle in the ganglion, of then injecting a few minims of novocain solution, with the result of completely anesthetizing the areas of distribution of the nerve. The alcohol was then injected without pain, but with the disappointing result that after a few hours "the face woke up", and the pain returned as before. Only one explanation can be given for this, and that is, that the solution of novocain deposited in the ganglion diluted the subsequent dosage of alcohol to such an extent, that it was no longer powerful enough to disintegrate the ganglionic structure.

Before starting the injection of the alcohol, the patient should be placed in that position in which it is easiest for the operator to visualize the structures to be penetrated by the needle (Fig. 7). The same position of the patient should be maintained in every case, for it is only in this way the operator may finally acquire a satisfactory technic. The writer prefers a horizontal position on the table, which is raised to such convenient height, that the surgeon is in no sense hindered in passing the needle. The head is so placed that the body of the mandible stands perpendicular to the table when the mouth is closed (Figs. 4 and 7). An assistant who knows the anatomy and the essential points of the injection thoroughly well, and who directs the angles at which the

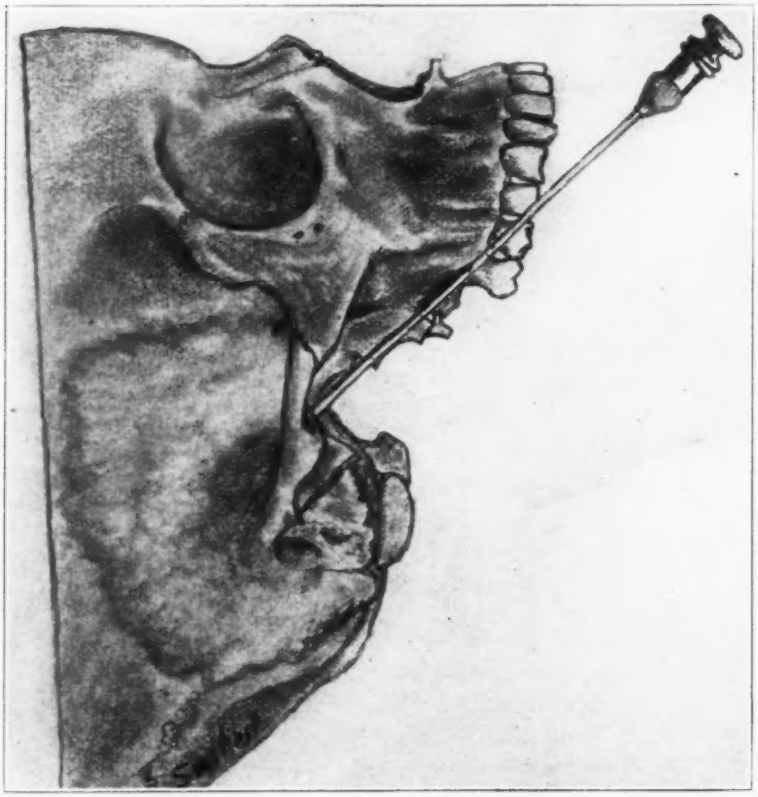


Fig. 4. Shows proper position of the skull, angle of the needle and relationship of the needle to the molar tooth, alveolus and the pterygoid process when the needle is entered into the foramen ovale.

needle be held, is of inestimable value in quickly and successfully passing the needle to its proper destination in the ganglion.

Operators very generally enter the needle at some point on the face and pass it to the foramen ovale through the tissues of the cheek. The writer early chose a point of entrance in the mouth, and believes it best. A 10 cm. needle with stylet, beveled at the point, and graduated from the point up to 6 cm. is used. The needle is entered in the furrow of mucous membrane between the aveolar process of the upper jaw and the cheek, at a point opposite the forward border of the last molar tooth, the needle penetrating the tis-

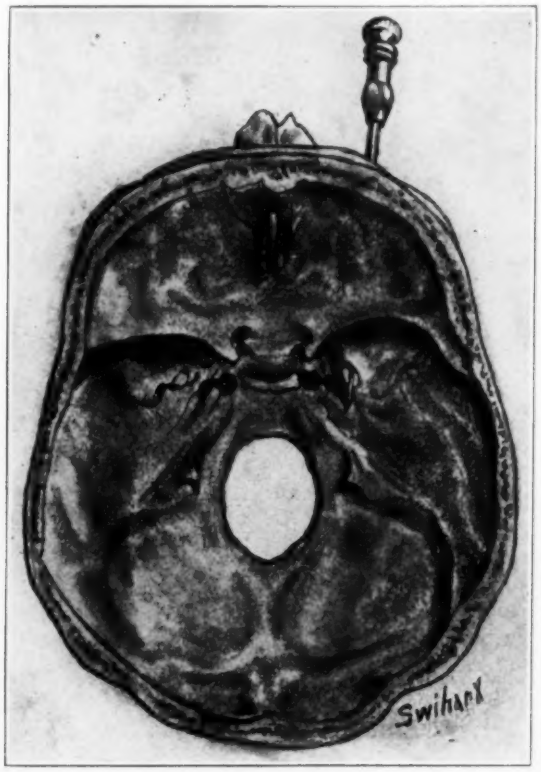


Fig. 5. Shows point of needle after it is properly passed through the foramen ovale in a position where it would penetrate into the heart of the Gasserian ganglion. Exactly the same position of needle as shown in Fig. 4.

sues at an upward angle of about 45° and an inward angle, toward the median line, of from 5 to 10 degrees (Figs. 4, 5, 6 and 7). The needle, with its stylet in position, is pushed gently, maintaining the above angles throughout to a depth of 4 cm. or perhaps a little more, when, if the position of

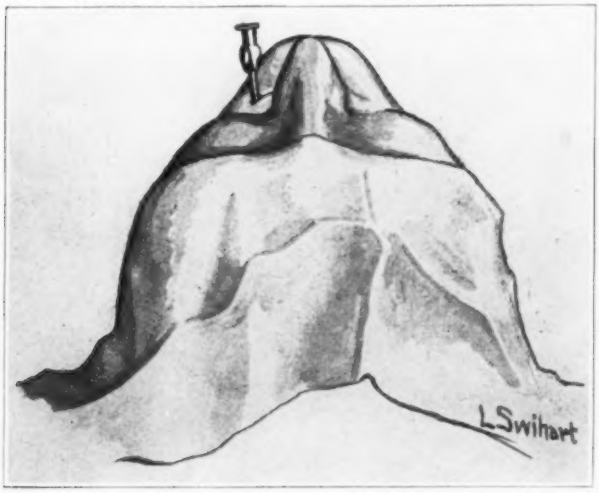


Fig. 6. The actual patient in normal position for ganglionic injection. The needle has been pushed through the foramen ovale into the heart of the ganglion. Note the angle at which the needle stands.

Fig. 7. Shows the proper position of the patient's head on the table and the upward angle the needle must take to reach the foramen ovale.

the patient and the angles of the needle are faulty, bone will be encountered. In case the position of the patient on the table is correct and the needle is passed at proper angles, the needle encounters the osseous rim of the foramen ovale at once, and may then be carried immediately to the required depth in the ganglion, the whole procedure lasting but a few seconds. Should one be not so fortunate, both operator and

Fig. 8. Showing the floor of the middle fossa. External to the foramen ovale is a large angular elevation of bone. To detach the dura from this angular osseous mound would require skill and patience. Also shows the osseous opening in the skull through which the operation of root resection is made.

assistant then make accurate observation of the postion of the patient's head and of the angles at which the needle stands. When needed corrections of faulty positions are made, the foramen is easily entered if the same be normal in every way. Should further difficulty in entering the foramen be experienced, the same would be evidence of an abnormally placed foramen, in which case the operator must manipulate the position of the point of the needle until the opening is found and entered.

How may the operator know when the needle has entered the foramen?

1. By the depth of penetration of the needle, which usually is not more than 4 or 4½ cm. It may be as shallow as 3½ cm.

2. By the feeling imparted to the hand of the operator as the needle enters. There is resistance to the point as it penetrates the denser tissues which fill the foramen. This feeling cannot be described, but will be acquired with certaintly after numerous injections. When once acquired it is an invaluable aid.

3. The patient, although apparently oblivious to all about him, from the narcotic, will suffer such agonizing pain, that he makes sharp outcry as the needle enters and penetrates the trunk of the nerve.

4. The angles at which the needle stands are observed and found correct.

5. The needle may be pushed to a further depth of 1 or more cm. without meeting obstruction.

When it is believed that the needle has of a certainty entered the mouth of the foramen ovale, and has of course pierced the sheath of the third division of the fifth nerve, the alcohol may be injected, provided only this branch is affected by the major neuralgia. The stylet is withdrawn, it is noted whether or not blood or cerebrospinal fluid escapes from the cannula, and if either does not, 10-15 m. of 95% alcohol are slowly injected, using a proper syringe. Force must never be used in the injection of the alcohol either into a nerve trunk or the ganglion, for the reason that if employed, spreading of the alcohol to distant and dangerous structures will more than likely result (Fig. 1).

If two or all divisions of the fifth nerve are involved in the major neuralgia, it usually is necessary to inject the gang-

lion itself. As already stated, to inject the ganglion may have dangerous consequences, and hence must be done after the best plan experience may dictate. The injection should not be made too deeply into the ganglion. No force should be used in making the injection, and the alcohol should be deposited as nearly as possible in the center—in the substance of the ganglion—and not at its periphery, where it might find lodgment in the arachnoid spaces and be diffused to distant parts. Therefore, having found the foramen ovale with the point of the needle, the operator must accurately note its depth as indicated by the centimeter marking on the needle. He may safely push the needle to a greater depth, but under no circumstances should he inject alcohol at a greater depth than 1 cm. from the foramen.

It may be asked, how is one to know that the point of the needle is at or near the heart of the ganglion, rather than in the arachnoid spaces near its periphery? This is reasonably well proven by the fact that the needle may be passed further into the ganglion without meeting obstruction, after the point has passed the foramen ovale, for, should the position of the point of the needle be faulty, it will pass through the ganglion at this depth and osseous obstruction will be met, in which case it would of course be known that the needle is not in the ganglionic center. Here, again, a valuable sense of touch is acquired by experience, which no description will suffice. In case, therefore, the needle has been pushed 1 cm. beyond the foramen ovale without meeting any obstruction, it is reasonably certain that it is properly placed. It should, therefore, be slightly withdrawn before any alcohol is injected. Six or eight minims are slowly deposited in the central part of the ganglion. Withdrawal of the needle is then made, and an equal amount of alcohol is injected every 4 mm. until the foramen is reached, at which point the final amount is expressed.

After finishing the injection, the needle is left in place for a few minutes, in order to determine the effect of the alcohol on the surface endings of the nerve. In case "a center hit" has been made, numbness of the face, forehead and mandibular regions soon is manifest. This fact will of course be difficult to determine if the patient is too completely narcotized, and it is because of this fact that a large experience in the preparatory narcosis of the patient is valuable. If too

lightly narcotized, so much pain is produced by the injection, that the patient is restless and hence greatly interferes with properly carrying out the procedure; if too deeply narcotized, one is unable to know accurately what the result of his efforts may be. However, after some experience, the surgeon is usually able to know a few minutes after the injection is made whether or not he has succeeded. If he has not, he may make further injection at that time, or he may await another day. If the patient is too deeply narcotized to answer questions, it is safest to wait.

The patient should be kept in the hospital under observation and treatment for one or more days. Should the first injection prove wholly a failure, it may be repeated next day. Should partial relief result, one may wisely await the return of the former pain before a repetition. When all three branches of the nerve are anesthetized completely by the injection, the cornea of the corresponding eye is of course wholly insensible. In such instances, the patient may be injured by the subsequent lodgment of foreign material, or by the patient himself rubbing the eye to the point of injury without realizing the fact. The eye should therefore be protected from injury and trophic changes by the application of sterile olive oil, the instillation of atropin, and by a carefully adjusted eyepad. Colored glasses should be worn, and the patient should be warned to avoid bright lights, dust and other sources of eye injury.

Other injuries resulting from alcohol injection, such as facial and ocular muscle palsies, may be greatly minimized by following the rules here outlined, especially those relating to injection without force, and reasonable assurance that the point of the needle is in the substance of the ganglionic tissue, and not in or near the arachnoid space when the alcohol is injected.

Results.—To be wholly successful, the alcohol injection must be made directly into the nerve tissue. If deposited outside this tissue and merely in the vicinity of the nerve, only partial and transient relief may be expected. If the nerve structures are "squarely hit", complete anesthesia of long duration occurs in all cases, and in all areas of the corresponding side of the head supplied by this nerve. Complete relief from the major neuralgia at once results. No one believes that this relief is ever a permanent cure, and the duration of cessation

from pain varies very much. Beckman states that the average period in 120 of his cases was 9.4 months. His longest case was 5½ years. Beckman's experience in this respect seems to coincide with that of others. Patrick says that good injections last from 6 months to 4 years. The writer has one case that has continued well for nearly 6 years, and it is not uncommon to have no return of pain in three or more years.

The first injection, or even a second or third, may prove ineffective in any case. All writers attest this fact. Adson states that the average number of injections given per person at the Mayo Clinic was 3.5, while the average period of relief was 7.7 months. 2 cc. of 80% alcohol were used by Adson in each injection.

Patrick, in a report of 500 deep injections into the second and third branches, states that there is always great uncertainty as to whether or not the operator may hit the nerve squarely, and claims only 18% of successes at the first injection. This author states that a good injection lasts from six months to four years.

Undoubtedly the period of relief depends on two factors, namely, the strength of the alcohol used, and the degree of success attained in injecting it directly into the nerve trunk or ganglion. There can be little doubt that 80% alcohol is too weak, and that, therefore, alcohol as nearly 100% as can be obtained should be used. It is a mistake to inject a solution of novocain into the nerve or ganglion to numb it before finally injecting the alcohol. The novocain solution dilutes and makes ineffective the alcohol, and for that reason should not be used.

Alcohol injections have a maximum effect only when injected directly into the nerve structure. It is unreasonable to expect relief from pain for any considerable period if the injected alcohol is lodged only in the vicinity of the nerve, is merely infiltrated about the outside of the nerve, and not deposited directly into its substance. Patients who have been relieved for periods of five years were undoubtedly those in which a perfect injection, a "center hit", of the nerve or ganglion had been made. Cases in which relief lasted but a few weeks, are, on the contrary, those that were imperfectly treated. The difference does not lie in the patient or in the severity of the disease, but solely in whether or not the injection was properly made. Acquirement of skill in hitting the

nerve structure squarely is, therefore, the first essential to success. It is undoubtedly possible, through anatomic study and large experience, to inject the medicament successfully into the nerve structure, and not merely into its vicinity, in a large proportion of cases. When this is done, the average period of relief will be very much longer than at present, fewer unpleasant after effects will be recorded, and fewer major operations will be required.

Treatment by Surgical Methods.

Authors of text books and surgical writers in general advocated, until recently, methods of treatment of tic douloureux by resection of peripheral nerve trunks and avulsion of as much of the central part of the trunk as possible. Elaborate directions were given for following the several divisions of the trifacial nerve from some superficial position up to the foramen of exit from the skull, where the branch was finally twisted away. The writer once presented this subject, giving a detailed description of methods and tabulated results. Longer observation of cases thus operated has convincingly proven, that all surgery directed solely to severance, avulsion or other means of destroying the peripheral branches of the trifacial nerve, is ultimately unsatisfactory as a means of cure for major neuralgia, because the former pain sooner or later returns in its original severity.

There is reason to believe that the pathology of major neuralgias, whatever its nature may be, is in the Gasserian ganglion, and that, therefore, any method of treatment which does not attack this ganglion or its sensory root must end in failure to cure. Leading surgeons now recognize this fact, and confine their efforts either to removal of the ganglion or to section of the posterior root. Some English and Continental surgeons still attack the ganglion, while American operators, led by Cushing, Frazier and Adson, advocate section of the root. Section of the root undoubtedly is the operation of choice. It accomplishes all that any operation can accomplish—permanent cessation of pain—and its execution is easier and vastly safer than subtotal removal of the ganglion.

Section of the Posterior Root. Since this procedure requires entrance into the cranial cavity, absolute sterility is essential. The hair is shaved from above and behind the attachment of the auricle for a distance of 3 inches. This

area, the face and adjacent temple are given the ether, iodin and alcohol sequence. The area thus sterilized is shielded from other portions of the head by sterile drapings fastened to the skin with clamps. Morphin, administered hypodermatically three-fourths hour previously, is helpful. Ether is given by one thoroughly trained in the art of its administration. The patient may sit upright in a chair, as advocated by Frazier, or may lie prone on an operating table. The upright position has the advantage that less anesthetic is required, less bleeding occurs, and, what is of more importance, the operator can with greater certainty at all times recognize the anatomic structures and relationships with which he must deal.

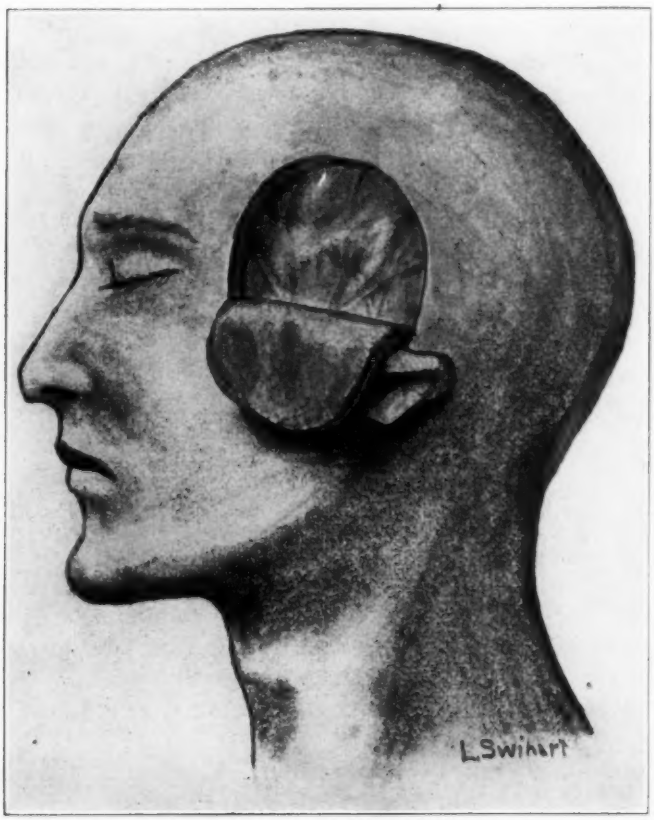


Fig. 9. Skin flap turned down. The illustration shows the incision too far toward the angle of the orbit, and slightly higher up than necessary.

The first stage of the operation is quite similar to the subtemporal decompression described by Cushing. The lines of incision should be so made as to avoid the upper or temporal branch of the facial nerve, since if this nerve be injured, impaired motion of the forehead and orbital angle results in noticeable deformity (Fig. 9). Frazier's plan meets all requirements, is easy of execution, and there is much less bleeding from the external wound than formerly, when the incision extended to the bone throughout its entire length and severed the superficial temporal artery. In stout pa-

Fig. 10. Shows the flap turned down, the temporal muscle and its aponeurosis partially severed from the zygoma and fully retracted so as to expose the field for removal of skull. The wound is shown larger than necessary.

tients, with heavy temporal muscle, in order to expose a sufficient area of skull to permit free manipulations of the operator, it is necessary to sever the attachment of the aponeurosis and temporal muscle from the zygomatic arch (Fig. 10). Formerly, the zygomatic arch was temporarily or permanently resected, but this is unnecessary, deforms the face and should not be done (Fig. 16).

When a sufficiently wide area of skull is exposed, the bone is perforated with a Doyen burr. This opening is enlarged to the size of a silver half dollar by means of a stout rongeur (Figs. 10 and 11). It is advantageous to bite away the skull downward as far as the subtemporal ridge, since, when this

Fig. 11. Shows the skull bitten away over an area large as a silver half dollar. The lower margin of the osseous wound is on a level with the floor of the middle fossa of the skull. The course of the middle meningeal artery is well shown.

is done, the operator is enabled to work along the floor of the middle fossa in a straight line—an almost invaluable aid to easy, correct methods of dealing with the problems of raising the dura, ligation of the middle meningeal artery and cutting the root.

When the osseous opening is complete, the dura is detached in all directions around its margin. When this is properly executed, the adjacent brain may then be lifted more easily and safely, conditions necessary to expose the deeper parts of the fossa in the region of the ganglion. The dura separates easily everywhere except on the floor, where it is somewhat firmly attached. In a considerable percentage of all skulls, there spring from the floor one or more osseous

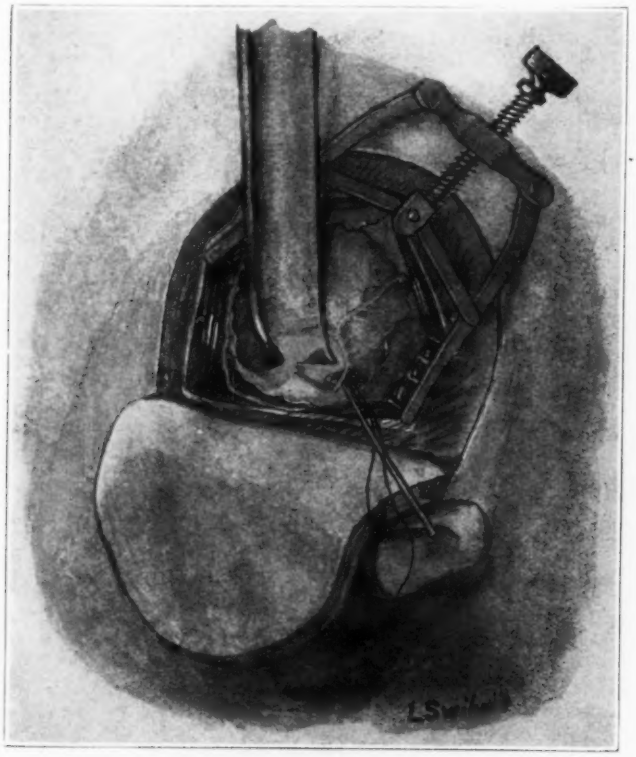


Fig. 12. The dura has been detached around the whole margin of the osseous wound. An electrically lighted elevator is shown lifting the dura and brain away from the floor of the fossa, and putting the middle meningeal artery on the stretch. A ligature carrier is passing behind the arterial trunk as it emerges from the foramen spinosun.

mounds, sometimes of considerable size, and to detach the dura from these, when present, requires patience and skill, since otherwise the dura may be torn, and the whole cavity at once be flooded by cerebrospinal fluid (Fig. 8). The middle meningeal artery also follows the pathway along the floor of the skull, and carelessness in elevating the dura may tear this vessel and result in troublesome hemorrhage.

Following the detachment of the dura, the brain may be elevated sufficiently to expose the deeper parts. Electrically lighted elevators are convenient for this purpose. When the brain is lifted and the depths lighted, the surgeon notes the course of the middle meningeal artery, and follows it from without to its point of entrance through the foramen spinosum. Further elevation of the brain will pull the artery somewhat from its foramen, which is helpful, but still greater elevation will likely rupture the vessel (Fig. 12). The artery

Fig. 13. The middle meningeal artery has been ligated. The dura mater has been elevated to the ganglionic posterior root, and the brain is lifted so as to expose the third division and ganglion.

must therefore receive attention. Frazier plugs the foramen with cotton. Ligation is not especially difficult, is permanently safe and should be done. After ligation, the vessel is cut centrally to the ligature. This releases the dura, which may then be elevated to the superior border of the petrous portion of the temporal bone. The brain is then lifted sufficiently to expose the third division of the fifth nerve (Fig. 13). With the brain well elevated and the mandibulbar division exposed and in good light, the dura is separated from the dura propria, or the sheath of the ganglion. This step is not easy, for the two layers of dura are rather closely adherent, and the point of beginning the separation is not easy to determine. A moderately sharp elevator is used, and if the greatest care be not exercised, the dura may be punctured, with the result that leakage of the cerebrospinal fluid takes place, to the great annoyance and delay of the operator. Cer-

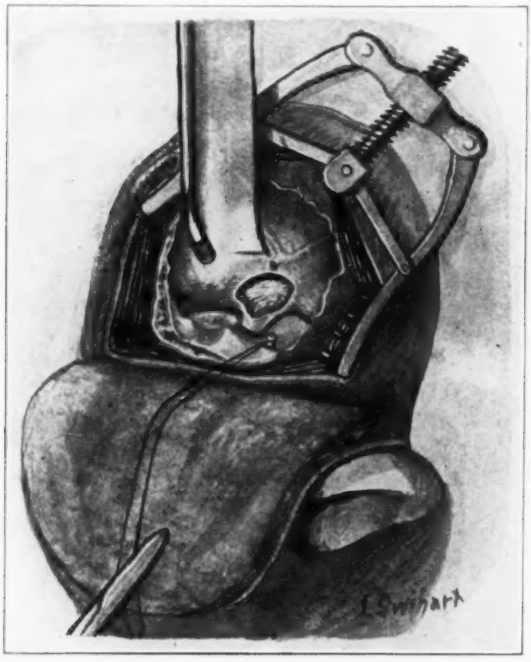


Fig. 14. Shows the dura propria separated from the dura, the dura propria incised and pushed aside so as to show the course of the ganglionic fasciculi running toward the posterior root. Note the great displacement of the dural end of the middle meningeal artery that is necessary to make a good ganglionic exposure.

tain lines on the dura that have been pointed out by some surgeons as serviceable landmarks in making this separation of the dura from the dura propria have not been observed by the writer, either in operations on the cadaver or on the living, and hence it is necessary for the operator to visualize his anatomy well, and to do the separation accordingly.

When once this separation of dura and dura propria is satisfactorily accomplished, the sheath of the ganglion is incised from below near the foramen ovale, upward and backward in the direction of the nerve fibers, to the point where the posterior root passes over the petrous portion of the temporal bone. When the sheath of the ganglion is thus incised and is further dissected from the ganglion and pushed aside, the fibers of the posterior root may be seen (Fig. 14). Cutting the posterior root is accomplished by passing a blunt right angled hook under the root and raising it so as to fully identify the structure (Fig. 15). Identification is not

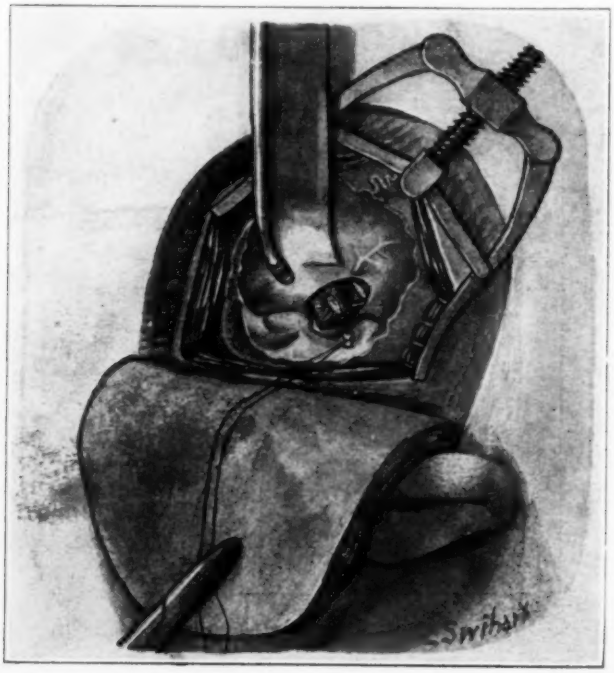


Fig. 15. Shows the posterior root pulled downward after section. Shows the motor root intact in its normal course.

difficult, since the position and appearance of the coarse fasciculi are at once assuring. In severing the sensory portion of the root, it is desirable not to cut the motor division, and to avoid doing so is not easy. The motor branch lies entirely under the sensory fasciculi, and runs in a horizontal direction from before backward to the foramen ovale. The two divisions of the root must be separated and each identified. The motor root is many times smaller than the sensory. It is a rounded cord, resembling any ordinary motor nerve. To separate and identify the two roots on the cadaver is not a simple matter, and is, of course, much more difficult at operation on the living. Frazier advocates the use of an electrode, which he applies to the portion he has isolated and believes to be the motor root, when, if the portion really is the motor root, the fact is evidenced by contraction of the exposed portion of the temporal muscle.

In patients whose ophthalmic branch is not affected by the neuralgia, some surgeons advocate leaving intact some of the most anterior fasciculi of the sensory root, thus avoiding anesthesia of the eye and its complications. Leaving these fibers is of course more or less hazardous, since there is no known plan by which the operator may be absolutely sure that he is not also leaving untouched some of the fibers to the second or third divisions, and since, should he do so, the patient will continue to have neuralgia after the operation. A further objection has been made to leaving intact any part of the sensory root, on the ground that not infrequently this branch of the trifacial, though not involved in the neuralgia at the time of operation, later becomes affected. It is, therefore, wise to sever the entire sensory root. Formerly, the central portion of this root was avulsed. Since eye complications have followed avulsion more often than merely cutting the root, avulsion should be abandoned, especially since complete severance is always followed by immediate and permanent relief.

Severance of the posterior root completes the operation with the exception of closing the wound. Any bleeding of consequence must be fully checked before final closure. The soft parts are replaced as nearly as possible into their original positions. Each layer is sutured separately with catgut. The skin is finally adjusted and sutured with silk or dermol. Since there is always some subsequent oozing from the depths of

the wound, a tissue drain is left in the lower angle, to be removed at the first dressing. Healing is complete within a week and the patient leaves the hospital in about 10 days.

Difficulties Met with in Root Resection.—1. Bleeding. Many small diploic veins supply the basilar dura of the middle fossa; hence, when the dura is elevated, these are torn off, and there is more or less venous oozing. This hemorrhage is, of course, seldom severe, but when occurring in the narrow

Fig. 16. Shows great deformity of face resulting from permanent resection of zygoma.

space available for the operation, it obscures the field and causes delay. If considerable, the foramina may be filled with bone wax, or as Frazier advises, may be rubbed or plugged with pledgets of cotton. Removing the brain retractor and allowing the brain to fill the cavity for a few moments will, by the pressure exerted upon the bleeding vessels, often be sufficient.

2. The middle meningeal artery or one of its branches may be ruptured in elevating the dura mater, especially along its course on the floor of the middle fossa. This can usually be avoided by care in the selection of an elevator that is not too sharp, and by proper caution in its employment. Should the vessel be injured, it may be clamped in an artery forceps and the branch ligated; or the main artery may be ligated at the foramen. The anterior branch of the middle meningeal artery sometimes enters an osseous canal in the skull instead of lying upon the dura as it does normally. In such cases, it may be accidentally bitten away with the rongeur in making the opening through the skull. Since there is no method by which it may be determined beforehand that the vessel lies in the bone, the fact that it often does so should always be borne in mind, and the osseous removal not made more widely than actually necessary toward the position of the vessel.

3. Injury to the dura. Injury to the dura may occur along the floor of the middle fossa, where it is closely adherent to the bone. In case the floor is rough and occupied by sharply angular, osseous mounds, it may not be easy to elevate the dura without tearing. When, therefore, a rough floor and closely adherent dura are encountered, one's patience may be tried, for should the dura be ruptured the wound will be flooded with cerebrospinal fluid, and much delay occasioned in finishing the operation. Injury to this membrane is most likely to occur when separating the dura from the dura propria. The lines of demarcation between the two, which should indicate the point of beginning such separation, are not very clear in any case, and the operator must often be guided by other landmarks, especially by the third division of the nerve, in its course from the foramen upward toward the ganglion. Separation of the dura from the ganglion has proven, for the writer, the most difficult part of the whole operation of posterior root resection. Strangely, no anatomist

has as yet given an adequate description of the dural relationships to the ganglion, and many dissections of this region have been necessary to give the writer a fairly satisfactory working knowledge.

4. Mortality. The percentage of recovery from operations on the Gasserian ganglion has had a marked upward trend in the past 30 years. In 1895, there was, according to the tables compiled by Tiffany, a mortality of 22.5% of all operated cases, while in 1909, in a series of 200 such cases, the percentage was 3.5. In 1921, Frazier reported 157 operations with but one death, a mortality of .6%. Adson reports having performed 116 root resections with three deaths, a mortality of 2.6%. The writer has had one death in 16 cases, a little more than 6%. This death occurred from pneumonia in a man of 75, one week following section of the root, and should, perhaps, not be charged to the operation.

Results.—Spiller asserted, in 1901, that if the posterior root of the ganglion were cut, union would not take place and that, therefore, the pathway of sensation from the fifth nerve to the brain was forever broken. Experience in severing the sensory root in many cases since has proven the truth of Spiller's discovery. The result, therefore, of completely cutting the posterior root of the ganglion in any patient suffering from major neuralgia of the fifth nerve is a permanent cessation of the neuralgia. This happy outcome is so complete and sudden, that it is amazing alike to patient and friends. The patient who was unable to eat, drink, shave, wash or touch the face without the likelihood of a neuralgic "strike", and who lived in constant terror of bringing on the pain, suddenly, almost as if by magic, is completely relieved, and can do as he dared not do before. Occasionally a patient is operated either by injection of alcohol or by root resection who, on first experiencing the complete numbness of the face, seems greatly disappointed at the result, even though entirely free from pain. Several of the writer's patients have said at first they were sorry they were operated. One, a glass blower, fancied he should be wholly unable to continue his trade. All these soon have forgotten in great measure the annoying anesthesia, and ultimately express profound gratitude.

So effective are the means of cure that may at present be directed against major neuralgia, that all who suffer may be promised relief at but a minimum risk to life.

REFERENCES.

Adson, A. W. Journ. Am. Dental Assn., 1922.
Adson, A. W. Collected Papers Mayo Clinic, 1919.
Beckman, E. H. Annals of Surgery, 1916.
Barnhill, John F. Trans. Am. Rhinol., Otol. and Laryngol. Assn., 1919.
Cushing, Harvey. Journ. A. M. A., Aug. 14, 1920.
Flesch, Berthold. Wiener med. Wochenschrift, 1914.
Frazier, Chas. H. Keen's Surgery Vol. IX.
Frazier, Chas. H. Journ. A. M. A., 1921.
Frazier, Chas. H. Journ. A. M. A., Jan. 8, 1921.
Kanavel, Allen B. Address before the Muncie Academy of Medicine, 1924.
Martin, H. H. Southern Med. Journ., March, 1914.
Martin, H. H. Southern Med. Journ., April, 1915.
Martin, H. H. Journ. A. M. A., 1919.
Patrick, Hugh T. Journ. A. M. A., Jan. 20, 1912.
Patrick, Hugh T. Journ. A. M. A., Dec. 4, 1909.
Spiller, W. G. Keen's Surgery, Vol. II.
Sachs, Ernst. Annals of Surgery, 1917.
Sharpe, William. Annals of Surgery, Vol. LXVIII.
Vaughan, George Tully. American Surgery, 1917.

DISCUSSION.

Dr. Joseph C. Beck, Chicago: It is not sufficient to mention these two types, because there is another type which exists only in the mind of the individual. It is the psychic type, always the most difficult to deal with. A psychiatrist or neurologist is necessary to make a diagnosis of this type. We are the ones that are interested in the minor neuralgias, especially those which we have classified as induration neuralgias, that is, the type which follows multiple operation on the nasal accessory sinuses, with headaches which are so difficult to contend with. The major neuralgias, or true tic, that have been described by the essayist, are of course as easy to recognize as a patient with renal colic and stones in the gall bladder.

Doctor Barnhill said he would inject the third nerve; I am sure he meant the third branch of the fifth nerve.

In injecting the ganglion, it is not possible to prevent neuroparalytic keratitis in every instance. The nerves are so distributed, that you may have a herpes zoster to a greater extent of the forehead. We had one such case that Doctor Gradle saw with me, and while the girl has her eye, the vision is reduced.

It is true that a good injection of alcohol in the ganglion will destroy the substance of the ganglion for a longer or shorter period, but, unfortunately, the anatomy is quite difficult to understand. The sheaths or layers of the dura will protect the ganglion, so that a complete medication will not take place. But most of these injections are made in old people, and as a rule they die before recurrence of the pain. I have a case that I injected eight years ago, an old lady, and she is cured of her major neuralgia; but in most of the cases, injections are not successful, and a posterior root resection is the thing to do when the patient suffers as much as these patients do. Go down and sever the root, as Doctor Barnhill says, and they are cured forever. Why not cure forever instead of trying to medicate and procrastinate? When your patient is so ill, it is my idea that that is the thing to do.

NONREFRACTIVE ASTHENOPIA.

WILLIAM THORNWALL DAVIS, M.D.

WASHINGTON, D. C.

By this title is meant asthenopia *not* due to refractive errors and muscle imbalances.

The derivation of the word asthenopia signifies weak eyes, and this is my understanding of its present definition. When we remember that a weakness of any function of the body may result from an almost infinite number of causes, we are brought to a realization that the whole science and art of medicine may be called into requisition when we approach this, at times, very difficult subject of asthenopia.

It is my earnest desire to invite your attention briefly to a few of the chief etiologic factors in the production of asthenopia not due to ocular causes.

There is nothing new in what is to follow, but repetition may serve to fix in our minds more firmly certain essentials that may be of help in refraction, that part of our work comprising not less than 80% of the whole, which appears so easy and is in reality so difficult; that we do a great many years before we realize we know so little about it; that part of ophthalmology where "Fools rush in where angels fear to tread"; that science that is mastered in a 90 day course!

To those of us with less experience, the repetition of known facts correlated to advantage, possibly may be of value.

Jackson advises us that as we have no unit of suffering, we must not be misled by the patient's description of it. All who make a practice of carefully correcting errors of refraction, find a certain number of patients whose sufferings are not relieved by lenses and correction of imbalanced muscles. It is to this class of cases that the following remarks are directed. Doctor Jackson further remarks that pain is the tongue of disease by which effective protest is made against the overstepping of the limits of endurance by departure from conditions of healthy living. Careful analysis of the life of the asthenope will sometimes show deep lying, unobtrusive, but long acting causes. Any rehabilitation must be founded

on investigation and inference going deep and extending far back.

The writer recognizes such cases by the fact, that after proper correction of refractive errors or muscle imbalances has been given them, they return unrelieved. In some cases, they can be recognized in the beginning as having a deeper significance than the usual case of refraction; and in some cases, there will be no need for refractive or muscle correction.

Batten says what we now need is the ophthalmic physician rather than the ophthalmic surgeon. Further progress in ophthalmology will be mainly on medical lines, and will require men with a physician's training and experience. He says it is doubtful if the cure of glaucoma, detached retina and the like will ever come through surgery. What we require is ophthalmologists who do their own work and have to live with their ophthalmic failures.

Only in passing is mention made of gross disease of the nasal accessory sinuses, such as acute and chronic suppuration. These conditions are of first importance in their effect upon the eyes, and may produce stubborn and incapacitating asthenopia or serious disease of the ocular apparatus, the optic nerve in particular. Suppurative and like obvious diseases of these structures should be promptly recognized and eradicated; this has become a platitude and requires no further observation here. The difficult sinus cases for the ophthalmologist, and apparently also for the rhinologist, are those discussed at length by Sluder. When studying the causes of nonrefractive asthenopia and seeking to eliminate the sinuses, we cannot always be certain of the X-ray and other negative findings. I do not presume to attempt instruction to the rhinologist, but rather seek further information in these difficult cases.

Chronic inflammatory changes in the soft parts may bring about changes in the bones. These changes in locations traversed by sensitive nerves or nerves of special sense are important. It is not the specificity of pathology so much as it is the specificity of location that is important. Thus these changes occurring in such a manner as to occlude a nasal fossa, or close the ostium of a sinus, or impinge on a branch of a sensitive nerve may cause marked symptoms and a long train of consequences. Closure of the frontal sinus causes long standing, low grade headache, made worse by the use of the eyes; the symptoms may be *ocular only*. A negative pressure develops causing sensitive sinus walls.

The floor of the sinus is the thinnest part and is dragged upon by the pully of the superior oblique, causing pain. Tenderness to external pressure may be present—Ewing's sign. Narrow noses plus hyperplastic changes in the soft parts and bones cause this condition. The resulting disturbances are *limited to asthenopia*. If the anterior ethmoid is involved, the tender point is at the site of the ethmoid bone, and these patients complain of a feeling of sand in, and pain behind the eyes. Involvement of the posterior ethmoid and sphenoid may cause occipital, parietal, or frontal headaches, made worse by use of the eyes, due to the dragging upon the sensitive walls by the cone of muscles attached about the apex of the orbit. Contacts of parts of the lateral walls with the septum may or may not be responsible for such symptoms. Let us briefly refresh our memory of the very intimate neural interrelations of the nasal accessory sinuses and ocular apparatus. The nasal, or sphenopalatine, ganglion has for its sensory root a branch from the fifth nerve, the great sensory nerve of the head, eyes, oral and nasal fossae, which is peculiarly subject to reflex irritations. Its motor root is the great superficial petrosal from the seventh, and we remember the susceptibility of this nerve to reflex irritations. The sympathetic supply of this ganglion is the great deep petrosal from the carotid plexus. The nasal ganglion supplies the nasal and oral fossae and sends branches to the sixth nerve, the ciliary ganglion and the optic nerve or sheath.

Our attention is arrested by the possibility of reflex irritation, particularly by the connection of the nasal with the ciliary ganglion and the common sensory supply of each. The only wonder is we do not have more reflex symptoms. The relations of the nasal ganglion vary and are important; it may be submucous to the nose and in intimate relations with the walls of the sphenoid, maxillary and posterior ethmoid cells. The lack of any regular anatomic arrangement of these cells makes them *always* an unknown quantity; too great emphasis cannot be laid on this fact. To appreciate this fully, we must thoughtfully read Schaefer's investigations of the subject. The sphenomaxillary fossa may, in effect, be considered a paranasal cell, not closed externally and filled with connective tissue. The nasal ganglion is in as intimate relation with the nose and paranasal cells as the optic nerve. It is reasonable to suppose it to be as subject to injury from nasal or sinus involvement as the optic nerve.

Sluder ascribes the redness of the eyes and epiphora, with

prominent eyes, photophobia and scotoma scintillans in an acute coryza, to the involvement of the nasal ganglion, and says these symptoms may be relieved by cocainization of this ganglion. Where there is chronic involvement of this ganglion, there may be a low grade asthenopia and conjunctivitis; and this, from the ophthalmologists' viewpoint, is pregnant with meaning in explaining some exceedingly difficult cases, etiologically, of nonrefractive asthenopia. Indeed, the great difficulty is to eliminate this possibility before one goes on to other investigations. Chronic sphenoidal inflammation may cause a third nerve involvement with obstinate asthenopia, which is apparently unexplainable from nasal examination. Some of these cases show a paresis of the superior oblique.

Nerves in bony rings, such as the optic and the third division of the fifth, are very prone to inflammatory involvement. These slowly progressing, hyperplastic bone changes cause chronic, slowly developing symptoms, the etiologic factors being very difficult to locate. If I may be permitted to diverge for an instant, I might say it is becoming my belief that all eye muscle imbalances are but symptoms, the relief of which should be sought by removing the cause rather than by treating the imbalance per se.

Morgan says faulty pressure in the nose will cause asthenopia; a moderate pressure of the middle turbinate will impair the function of the ciliary muscle. Bordley states that sinus disease is extremely difficult to diagnose; for some cases every scrap of evidence is needed. There may be slight interference with the accommodation or complete paralysis of this function. Nasal ganglion neuritis may also cause pain in the root of the nose, made worse by using the eyes and also on attempting the use of pinch glasses.

We sometimes find diseased tonsils causing asthenopia. This may operate in several ways: by reflexes, particularly when there are adhesions and evidences of long continued or successive attacks of inflammation of the tonsils; by chronic intoxication, causing reduced health with asthenopia; or by direct action of the poison on the eye itself, causing low grade uveal involvement with but little evidence of its presence save chronic eye discomfort on attempting near work. Likewise, these poisons may affect the nasal accessory sinuses, contributing indirectly to their inflammation or hyperplasia. Indeed, it is well known that diseased tonsils may be the starting point of diseased conditions in the body which, reacting upon the ocular apparatus, may cause

chronic asthenopia. One might mention cases in point, but it is unnecessary. The practical and important point is to recognize a diseased tonsil from one not diseased. Patients should not be subjected to tonsillectomy unless it is distinctly necessary.

Adenoid growths in the pharynx predispose to chronic asthenopia; their mode of action is well known and understood. They also predispose the patient to disease of the sinuses and pulmonary tract and greatly reduce his resistance and vitality, which indirectly affects the ocular apparatus.

A case of obstinate asthenopia as the only symptom of a chronic nonsuppurative ethmoid sinus might be mentioned. A man in the early thirties, an athlete and one of our most noted prewar aviators, was unable to use his eyes for near work. A minute study and observation over a period of two years revealed nothing to account for it. Examinations of the visual fields on numerous occasions were normal. He mentioned one day that while flying he noticed a dark object flitting before him in the sky or clouds. Finally, a minute paracentral scotoma in the right eye was demonstrated, being present only occasionally. Opening of the anterior ethmoidal sinus cured him permanently. There was no evidence of nasal or sinus disease other than the ocular symptoms of asthenopia and scotoma.

The writer has observed no case of asthenopia caused by ear disturbances. There is sufficiently close neurologic relation, however, for this to occur, and it is hoped in the discussion such cases may be mentioned. We are familiar with the nystagmus produced by the stimulation of the semicircular canals, and von der Hoeve invites our attention to an otogenic pupillary and palpebral reflex, and observes that the congenitally deaf and dumb show forms of pigmentary degeneration of the retina. Lowman mentions reflex symptomatic disturbances of the eyes from the cervical sympathetic plexus, which he believes to be due to reflexes via the labyrinth.

Disturbances of the sex organs produce a most profound effect on the nervous system; this is mostly observed in the female. The writer has frequently observed neurotic asthenopia, having for its basis uterine or ovarian displacement or disease. Those who have had the menopause prematurely precipitated are particularly prone to this. Sex trauma in early life is the occasional beginning of a neurosis manifesting itself in the disturbance of ocular function. The effect is undoubtedly a sympathetic nervous system disturbance. A case in point—the wife of a physician,

who was never able to become a mother, though having an ardent desire for children; the cause a contracted pelvis. She cannot use her eyes, and a searching study reveals no other cause for her symptoms. It is not easy to eliminate sex disturbances as an underlying cause of a chronic asthenopia. Many of these cases look upon the oculist as one who should go into only the ophthalmologic side of their condition; but it is our duty to comport ourselves as physicians and not oculists only, if we are really to hold the high place that we should in the confidence of our patients.

Byers mentions the establishment of menstruation and the cessation of this function as producing asthenopia, which passes off after the condition has become habitual.

In the case of a boy of sixteen with persistent asthenopia for two years, there had been numerous refractions. A medical survey showed him normal. Investigation disclosed excessive masturbation; explanation and suggestion promptly cured him.

Bond would explain the disturbed nerves of the female at menstruation as being due to upset endocrine function, and no doubt this plays an important part. In pregnancy, we may have greatly disturbed ocular function, but this is mostly of toxic origin and the method of relief is obvious.

Endocrine disturbances are the fad in medicine at the present time, and while recognizing their great importance, it is well to observe facts and not fancies. We certainly can do no better than to follow Fuchs, who says the clinical reports of cures by the use of gland substances are countless in number but carry with them no exact information. Stewart says "The contrast is great when we leave the desert where physiologists and experimental pathologists have wandered, striking many rocks but finding few springs, and pass into the exuberant land of clinical endocrinology, flowing with blandest milk and honey almost suspiciously sweet." Those ocular troubles which occur so often that their relation to glands may be said to be ascertained are but very few. "The eyes are only exceptionally affected directly by the alterations of the internal secretion of the ductless glands."

Persistent asthenopia is seen in cases of hyperthyroidism, and may be the first *apparent* symptom. In difficult cases of chronic asthenopia, it is the writer's custom to have a basic metabolism test. I have seen no cases as the result of hypothyroidism, but from the general asthenia in such cases, it would seem symptoms of eye discomfort might arise from the reduced

nerve tone affecting the accommodation and the visual act. No such cases have been reported so far as I know.

The thyroid is supposed to stimulate the thymus, adrenals and gonads, and to inhibit the pancreas, hypophysis and parathyroids. It stimulates body metabolism and destroys body toxins. It has an important effect on salt metabolism. The parathyroids are supposed to inhibit the thyroid, and to affect calcium metabolism. Destruction of the parathyroids causes tetany. There occurs ciliary spasm in tetany, which is of interest in connection with a possible cause of some cases of chronic asthenopia. We remember that convulsions in tetany produce cataracts in children, adults, and in pregnancy.

No direct action on the eye occurs from pituitary secretions. The pressure symptoms of pituitary tumors are characteristic; the subjective sense of blurring occurring early in these cases produces the effect of an asthenopia. These cases are very misleading and may be overlooked.

As to the gonads, Fuchs observes that the influence of sex cannot be denied; but it is as yet impossible to tell whether the predisposition of the tissues of the eye to certain diseases is due to direct action of the internal secretion of the gonads, or is affected by the intermediary of other factors.

Interference with the pancreatic secretion affects the eye indirectly from diabetes. This disease has much influence upon the eye, its nervous supply and tissues. The writer has seen a number of cases of most persistent asthenopia caused in this way; and in several, for some time it was the only apparent symptom. It would be well not to confine one's investigations to the urine only, but to determine the blood sugar. One case is worth mentioning. She was unable to use her eyes even for a few minutes; this persisted over a number of years, and exhaustive study failed to reveal any cause; she is the wife of an officer of the army and passed from observation for several years, returning after our knowledge of blood chemistry and insulin had come into existence. A high blood sugar content gave the clue, and under proper treatment her diabetes is under control, and she now uses her eyes ad libidum.

Steyn says slight changes in the retina, occurring as striation or a depigmentation area near the fovea, with asthenopic symptoms, may occur in connection with abnormal basal metabolic rate of endocrine origin. This frequently is accompanied with general and ocular fatigue and subnormal vitality.

Many of these cases have a hypometabolic rate and low blood pressure.

Fridenberg suggests that the hyperthyroidic eye tends toward subnormal accommodation, accompanied by various forms of asthenopia, and Zentmayer says asthenopia may be a prominent symptom in athyroidism. He speaks a word of warning as to linking obscure symptoms with endocrine disturbance.

De Schweinitz mentions the subjective blurring of pituitary disease, which may be present for several years before the condition becomes manifest. He suggests that testing the light sense might show it to be deficient.

The scotomata and partial or complete hemianopsias associated with pituitary disease may cause disturbances of vision *expressed by the patient* as asthenopia; this may be overlooked if field tests are not made.

One is impressed with the suffering of those with the arteriosclerotic form, among the most persistent of asthenopias; often this is one of the principal features of the disease. The refraction in these cases seems to vary, and a proper correction is difficult to get. But no correction relieves the patient; and the glass that he can use today, tomorrow he cannot. They frequently come early to the ophthalmologist. The vascular changes in the retinal vessels are not always marked, and the diagnosis is not as apparent as it would seem. The writer has noted a peculiar and characteristic reaction in the eyes of such patients. They see clearly for a moment, and then there is blurring; there is difficulty in ascertaining within a diopter, more or less, just what correction is proper, so that one often has to go over the refraction several times on different days. I have thought this probably due to the low nutrition of the retina, ciliary cramp or spasm of the retinal arterioles. It is frequently associated with a delicate veiling of the fundus. The blurring while refracting differs from that frequently seen in neurotic individuals, in that it can scarcely be overcome, whereas the neurotic blurring is more easily overcome. Low grade uveitis may exist in these cases, with symptoms so slight as to be easily overlooked. The causes, results, and pathology of angiosclerosis are well known and obviously require no discussion here. I consider this condition, in its earlier stages, to be of supreme importance in the etiology of nonrefractive asthenopia. In these remarks concerning angiosclerosis, it is the early stages of the disease

referred to rather than that stage where the advanced vascular disease has made the individual a horror to himself and his family.

In Washington, there are many young women in office who lead very unhygienic lives, resulting in intestinal stasis and chronic digestive disturbances. They take no exercise, eat improperly, consume much tea and coffee and some of them use tobacco to excess; they often get insufficient rest, due to their social activities. The result is absorption of toxins from the intestinal tract, which often affects the visual act, so that they get persistent asthenopia without any very apparent cause. They tell you they are well; one goes into the history of their life and habits and finds no glaring faults.

There is another class of patients who suffer in the same way. Each year a number of officers of the Army come to Washington to the War College and to duty in the War Department. They are a class of robust, physically fit men, accustomed to the out of door life. They suddenly find themselves for the time being converted into office men, and the change in their life habits results in the condition above noted. Both these classes of patients were very puzzling for a time, because they seemed perfectly well, and a medical survey showed nothing abnormal. Many of these officers find themselves seriously embarrassed in their duties, due to their inability to use their eyes for close work. Proper hygiene of life, proper food, and exercise relieve the situation. This sounds simple, but in the cases of the girl clerks, they are not always frank, and one has difficulty in ascertaining their habits of life in order to suggest correction. Not infrequently, one finds some young woman underweight and anemic, with no bad life habits, but having no grinding teeth; so long as the front teeth look well she was not concerned with the fact that she has to remain on soft diet, hence she is undernourished and usually takes to large quantities of tea or coffee to whip her up for her work. It is my custom to study these cases carefully and correct the gastrointestinal fault by attention to the habits; should this prove insufficient, they are referred to a gastroenterologist.

Gastric ulcer, colitis, chronic appendicitis, cholecystitis or other chronic inflammatory disturbances of the abdomen and pelvis may be obscure causes. One case complained of the usual chronic asthenopia, with acute exacerbations every few

weeks. Investigation disclosed "bilious attacks" preceding the asthenopia. This proved to be a chronic appendicitis. As she had had a "bilious attack" the night preceding, examination revealed exquisite tenderness at McBurney's point; she was sent to a general surgeon who operated at once, in time to prevent rupture of the appendix. She had no further asthenopia. Sir William Arbuthnot Lane says the eyes are always affected in intestinal stasis, and afford a delicate indication of the degree of autointoxication. Vitamine deficiency in lesser degree produces marked asthenopia. Compression of the eyeball induces a number of reflexes involving the viscera. The oculogastric and ocular colic are of interest in this connection. The dysenteries of the tropics and subtropics induce intestinal conditions leading to chronic intoxication. Uncinariasis may be a cause. One persistent case of asthenopia from Virginia resisted all measures of relief; he had the peculiar fish like stare of those infected with hookworm; examination of the feces showed ova, and appropriate treatment was followed by relief of his asthenopia.

The ingestion of certain toxic substances will cause asthenopic symptoms. One case had difficulty in reading extending over a period of some months; after all clues had been followed up and the case was about to depart, I found she lived in a residence of the gaslight period, and had a bedside reading lamp with a leaky rubber tube. A new tube settled the difficulty. She was suffering from a mild monoxid poisoning, it was presumed. Another case was a worker in the analytic chemical laboratory of the Federal Agricultural Department. He was ready to give up his profession on account of long continued, severe asthenopia. Every study imaginable was made in his case. He was working in arsenic preparations. His hair and urine showed arsenic traces; he was suffering from chronic arsenic poisoning. Omitting his arsenic analyses stopped his trouble.

Tea tasters and tobacco workers also may be affected. Lead, methyl alcohol, anilin and the salicylates will produce such symptoms. In some countries, there is consumption of impure alcohol, causing chronic intoxications of various types, vascular, gastrointestinal and asthenic. This should be considered in obscure cases.

So much has been said on the subject of dental disturbances, that it is universal knowledge. Asthenopia may be

caused by septic or reflex dental conditions. One should ever bear in mind Deever's three T's, teeth, tonsils and tummy.

Kahn refers to the common innervation of the eyes and teeth by the fifth nerve, and reports many reflex disturbances of the eyes from this source. The reflex disturbances require more care in diagnosis than do the septic conditions.

Dental conditions rank high in order of importance in the etiology of nonrefractive asthenopia.

Gout is the etiologic factor in a small percentage of cases. The diagnosis is difficult, and may be arrived at only by careful analysis of the family, and previous history and life habits of the patient. Blood and urine chemistry is of importance. Several cases responded to antilithemic measures after several months, when every other possible avenue had been followed. Diabetes and the involvement of the eyes has been discussed. No case of asthenopia of obscure origin can be considered to have been carefully studied if the percentage of blood sugar has not been ascertained. There are cases with no diabetic symptoms running a high blood sugar; one may think of them as in the prediabetic stage with asthenopia as a prominent symptom.

Several cases have had the most persistent type of asthenopia as part of the symptom complex associated with gastroptosis and ptosis of other abdominal viscera. The diagnosis in these cases is quite difficult, and the patient is often sceptical that a symptom like asthenopia can come as a result, or be one of the symptoms of such disease. One case had sought relief from asthenopia for years, and had been thoroughly examined and exhaustively studied. The patient was referred to Goldthwait as a chronic of some type. She had a moderate degree of gastroenteroptosis and six months' treatment of this condition fully relieved her long standing asthenopia.

Very persistent asthenopia may be seen in incipient cataract, with or without actual opacities in the lens; in such cases, when it is best not to acquaint the patient with the diagnosis, we are placed in a difficult position. We cannot relieve him with glasses owing to the irregular astigmatism, and he is, of course, greatly disappointed over his inability to have eye comfort.

Byers says the nervous apparatus of the eye forms an appreciative part of the brain's bulk. It participates through

intimate connection with practically every one of the various cerebral functions. As all toxemias affect the central nervous system, it follows that asthenopic symptoms must frequently form part of the clinical picture of systemic disturbances such as confinement, major operations, the nursing period, emotional disturbances, grief and anxiety.

The onset of acute infections, as for example, typhoid fever, may be with an acute asthenopia; also the anemias from any cause, and from hemorrhage. Glaring lights and badly faced desks will cause persistent asthenopia, and it may be difficult to ferret out these factors. A young woman had a moderate, but long continued asthenopia, which numerous ocular examinations and glasses had not helped; her desk faced a window looking across the street at a large white building; changing her seat so as to place the flaring wall behind her relieved her.

Those whose duties require residence in the tropics are predisposed to asthenopia by the tremendous glare of the sun on water, sand and arid stretches of plain.

Elliot invites our attention to tropical asthenopia from glare and other factors in the tropics, such as poor food, insolation or malarial infections. Some years of service under the equator with our Army have made me endorse his views. Very persistent and incapacitating asthenopia arises from the life one is forced to lead. Col. Elliott ascribes the sunlight as a cause; it is undoubtedly a powerful factor, but I am inclined to believe the reduced nervous, mental and physical tone incurred is equally a factor.

In this connection may be mentioned the difficulty experienced by aphakics, which might be classed as a variety of asthenopia. In the normal eye, the retina is pretty well protected from the ultraviolet rays by the lens and media. In the aphakic eye, this protection is lessened and this may be a factor. The different dioptric arrangement, difference in the size of the retinal image, and hence in the retinal stimulus transmitted to the cortical visual memory center, all probably combine to produce the effect, so that the individual may test 20/20 and read Jaeger 1, yet is unhappy and dissatisfied, and has persistent inability to use his vision. In many of these cases, cortical reeducation is necessary before we can get our patient to use his eyes.

Encephalitis lethargica is frequently first seen by the

ophthalmologist. Often there is extraocular paresis, but some cases have complained of an acute asthenopia, and the diagnosis is not always easy. I have seen a number of cases with a most pronounced and lasting asthenopia as a sequel of the disease. The reason for this I have not been able to determine.

The ocular symptoms of tabes and taboparalysis may include asthenopia, and this should be borne in mind in obscure cases. A searching neurologic examination may be necessary to establish the diagnosis. Disseminate sclerosis occasionally presents this symptom to a marked degree, and the early diagnosis is difficult. Myasthenia gravis may likewise thus affect the ocular apparatus, and the diagnosis be in obscurity until the disease has progressed sufficiently for diagnosis. One case presented incapacitating asthenopia for five years, resisting all attempts at diagnosis, until the disease had progressed to the stage where this was possible. She died from paralysis of the muscles of the throat.

Cerebral tumor may also cause asthenopia, as also may disturbance of the spinal cord. The obscurity of the diagnosis may place us in a difficult position. Mental disturbances after surgical operations of any type likewise may leave persistent asthenopia as a residue.

Paralysis agitans, migrain and polyneuritis may also cause it. Posey and Spiller say the optic nerve is not a nerve in the same sense as the peripheral nerves, but forms part of the brain.

There are other diseases of the central nervous system presenting this symptom rarely. We know the difficulties presenting themselves in the diagnosis of the rarer neurologic disturbance, and to eliminate this in searching for the cause of a profound asthenopia is indeed difficult.

Congenital word blindness may be confused by school examiners and others for deficient vision, and attempts to force the child may cause quite serious ocular disturbance of an asthenopic type.

The malingerer who claims asthenopia of persistent degree as a symptom offers considerable difficulty; fortunately, in civil practice we do not encounter them often.

Night blindness may have an asthenopia as part of the symptom complex; the underlying cause of the condition should be sought and removed if possible. Occasionally, a

patient just entering into presbyopia, especially women, will develop a mild neurosis following the use of a cycloplegic, declaring they could see well before the drops were used, but not after. Occasionally, also, this will happen following the ophthalmoscopic examination, and result in a complaining patient with persistent asthenopia. From such as these, good Lord deliver us!

Spinal pathology bears an important relation to ophthalmology. Lowman says the principal local lesions which we have found associated with the eye and balance complex of the body are cervical rib, osteoarthritis in the cervical and cervicodorsal region, fibrositic deposits, scoliotic twists and curves, impingements of the posterior processes in cervical lordosis compensatory to anteroposterior spinal malalignment and minor luxations of the vertebrae. It is thought the eye conditions resulting from these disturbances occur through the irritation of the cervical sympathetic.

Muscle fatigue produces toxemia. Due to malposition, the muscles are put on undue and constant strain, resulting in overfatigue or spasm. Thus, a normal daily life may require so much overwork due to this condition, that exhaustion may occur and the ocular apparatus be affected thru nervous exhaustion, toxemia and reflex irritation, all from spinal malposition. In fact, the malposition may not be confined to the spine; one case is mentioned by Lowman where asthenopia was relieved by proper padding of the shoes for malformation of the feet. It is well to bear in mind how fatigue affects the act of vision. Fergus says the source of sight is the mixture of five different functions: (1) Form sense. (2) Reading sense. (3) Light sense. (4) Color sense. (5) Perspective. Visual sensations pass to the brain and are interpreted by the mind. Helmholtz says the impressions of the sense organs are signs to our consciousness, and it is left to our intellect to learn their meaning. The optic nerve itself is blind; it merely conducts sensations produced by the image on the retina. Hence, we understand what a complicated nervous reaction is vision, and it is not difficult to see how fatigue, exhaustion or toxins acting on the nervous structures concerned in the complicated neurologic chain can cause dysfunction, which may be manifested as asthenopia.

Jackson says the brain works with certain tools. These tools are especially the organs of special sense, and it is

important that the tools be kept in good order. Eyestrain and brain strain may go unrecognized for years, and finally the individual is arrested in his course by the giving out of the brain, or by retinal irritation becoming so intense that it can no longer be endured. The brain is talking with the tongue of disease. Hence the profound asthenopia may be the evidence of profound fatigue, the result of malformation or disease.

Chronic respiratory infections may thus produce dysfunction by means of chronic intoxication acting upon the elements of the neurologic chain or thru exhaustion. Tuberculosis manifestly is the principal one of these infections. The writer has observed several cases in which asthenopia was the most annoying symptom of the unrecognized tuberculosis, and was promptly relieved by putting the patient at rest and so reducing the intoxication.

The neuroses offer a difficult class of cases to eliminate as a cause of one of the most pronounced and incapacitating forms of asthenopia. Ocular neuroses are described in the text books variously under the heads of asthenopia of hypochondriac or neurasthenic origin; also as copiopia (nervous asthenopia), retinal hyperesthesia and painful accommodation. It should be understood that these names refer to a clinical entity which we may designate as a psychosis, having for its origin an emotional or other functional disturbance of the nervous system, which often presents asthenopia as its outstanding symptom. Here we are not dealing with material things; this part of our profession should be for the moment left behind us, and we must enter into the mental and spiritual life of the patient in order to help him. Lancaster has given us a valuable contribution in this work, as have Lunt and Riggs. It is a branch of ophthalmology requiring careful thought.

Chronic inflammatory disturbances of the conjunctiva may present an incapacitating asthenopia as their chief and almost only symptom. The conjunctivitis caused by the diplobacillus of Morax-Axenfeld is typical of such cases and often goes unrecognized, so that the individual has many glasses, loses teeth or tonsils, money and time, when a solution of zinc sulphat would have done the work. It would be interesting to know if others have had the experience of the writer in having seen a number of unrecognized cases.

BIBLIOGRAPHY.

Stegman. Abnormal Metabolic Rate. Jour. Mich. Med. Soc., Sept., 1921, p. 335.
McGuire. Diagnostic Methods in Refraction Work. Va. Med. Monthly, Oct., 1923.
Hogg, G. H. The Eye as an Aid in Diagnosis Elsewhere. Med. Jour. Australia, Mar., 1923.
Van der Hoeve. Relations Between Eye and Ear. Annals Otol. Rhinol. and Laryn., June, 1923, p. 571.
Byers, W. Gordon. Canadian Med. A. Jour., Nov., 1921.
Cary, E. H. Further Studies of Ocular Manifestations Resulting from Fifth Nerve Irritation So. Med. Jour., August, 1922, vol. xv, p. 657.
Lyle, D. J., and McCord, C. P. Eye Symptomatology in Occupational Disease. So. Med. Jour., March, 1922.
Fridenberg. Trans. Am. Ophthal. Soc., 1920.
Jennings. Focal Infection in Relation to Ocular Disease. Pa. Med. Jour., March, 1922.
Wilson. Autotoxemia in Ophthalmology. Brit. Med. Jour., November, 1921.
Lowman, C. L. Spinal Pathology in Relation to Ocular Manifestations. Am. Jour. Orth. Surg., December, 1918.
Fergus. The Psychology of Vision. Glasgow Med. Jour., March, 1921.
de Schweinitz. Concerning Some of the Ocular Interpretations of Disorders of the Pituitary Body and Their Nonsurgical Treatment.
Kahn. Eyes and Teeth. Jour. A. M. A., April 21, 1923.
Terrien. Ocular Disturbances in Affections of the Respiratory Apparatus. Paris Med. Jour., Jan. 27, 1923.
Green and Sluder. Control of Pain in Functional Hyperesthesia of the Ciliary Muscle. Jour. A. M. A., Nov. 24, 1923.
Danilopn. Oculogastric and Oculocolic Reflex. Annals de Médicin, Paris, Feb., 1922.
Elliot. Tropical Ophthalmology, 1920.
Jackson. Visual Fatigue. Am. Jour. Ophth., March, 1921.
Jackson. Asthenopia. Am. Jour. Ophth., March, 1921.
Bordley. Ocular Manifestations of Disease of the Paranasal Sinuses. Archives of Ophth., March, 1921.
Sluder, G. Headaches and Eye Disorders of Nasal Origin.
Morgan. Ocular Diseases from Nasal Accessory Sinuses. Am. Jour. Ophth., Sept., 1923.
Moore, J. Foster. Medical Ophthalmology.
Israel. Some Ophthalmic Manifestations of Disease of the Nervous System. Jour. A. M. A., Oct. 7, 1922.
Fuchs, E. Ocular Manifestations of Internal Secretion. Archives of Ophth., July, 1922, vol. li, p. 307.
Batton. The Ophthalmic Physician and the Advancement of Ophthalmology. Lancet, Nov. 29, 1919.
Cary. Further Studies of Ocular Manifestations Resulting from Fifth Nerve Irritation. So. Med. Jour., August, 1922, vol. xv, p. 657.
Lyle, D. J., and McCord, C. P. Eye Symptomatology in Occupational Diseases. Nation's Health, Chicago, Oct., 1922, vol. iv, pp. 613, 615.
Davis, Wm. T. The Interrelation of the Teeth and the Eyes. Dental Cosmos, Phila., 1915, L. V. U., pp. 769-774.

DISCUSSION.

Dr. Frank Brawley, Chicago: I shall briefly stress only three conditions. First is the symptom complex which I call the vacuum sinus headache. Sluder, Robertson and the speaker published a description of this phenomenon independently in the same year.

The patient gives a history of unilateral pain in and above the eye, of the frontal sinus type, and in and behind the eye, on motion of the globe, of the ethmoid type. There is photophobia, lacrimation and pain on attempting to use the eyes. The frontal pain is increased on stooping. The symptoms appear after a heavy meal, the ingestion of alcohol, the menstrual period or sleeping on the involved side. There is never any sinus secretion found, and during an attack, shrinking of the mucosa of the affected frontonasal duct or probing, permits air to enter, and the pain, lacrimation, etc. promptly subsides.

The second condition is illustrated by a case I had the pleasure of seeing with Dr. John R. Newcomb. Briefly, the eye findings were greatly lowered vision, to 6/30, in the eye involved, full fields but greatly enlarged blind spot. Severe pain in and behind the eye, and total inability to use the eyes for any close work. There was also a pseudoepilepsy. The condition grew much worse after a nasal operation, when I first saw the case. Suction stopped the pain. On operation on the sphenoid and posterior ethmoidal cells, a shallow sphenoid was found, confirming the radiographs, and a large perforation in the superoexternal angle of the posterior sphenoidal wall, with a protruding button of pulsating dura, around which pus was draining. The depth of the cavity was measured with a probe, and a slot cut with a small round bur through the posterior sphenoidal wall, along the line of the lowest point of the cavity. Drainage thus established ended all pain, and within a week vision had gradually returned to normal. The infecting organism was staphylococcus albus, or a quite different ending to the tale would certainly have been written.

The third condition is found in the early symptoms of some brain tumors, particularly those involving the brain stem. A case in point is that of a young woman who complained of photophobia and pain in the region of the inner canthus of the right eye. There were, also, pulsations behind the ears on sudden turning of the head. The eye findings were all normal except a low adduction, combined rotatory and horizontal nystagmus, difficult to elicit even on extreme outward rotation, and variable. The temporal halves of both discs were suspiciously pale. These findings, together with a slight unsteadiness in her gait, suggested multiple sclerosis, which was confirmed by two neurologists. Later the speaker had the opportunity to make complete vestibular tests, which, much to our surprise, indicated a lesion of the right brain stem above the inferior cerebellar peduncles, involving the posterior bundles and probably the right cerebellar hemisphere. Papilledema appeared. Because of conflicting opinion, a decompression was not done and the patient became totally blind. About a year later, at the Mayo clinic, an inoperable tumor in this exact area was found.

I believe it is a necessity for every ophthalmologist to collaborate closely with a competent rhinologist, a dentist and a diagnostician, who are in sympathy with this work and who examine the patient before them

with the eye picture always in mind. Casual consultations with men unfamiliar with the possibilities are worse than useless.

The ophthalmologist must acquire the habit of incorporating a thorough general history with his complete eye history, and only then will he develop a most important angle of his work.

Dr. J. Ivimey Dowling, Albany, N. Y.: A broad interpretation of the phrase "asthenopia not due to ocular causes" is sufficient reason for me to relate results obtained by intranasal surgery for the purpose of overcoming alternating strabismus of both convergent and divergent types.

Dr. Lucien Howe intimates in his classic treatise, that there is a belief in the causal relationship of the nose to muscular insufficiencies. The question is, however, mooted, and it remains for the rhinologist to furnish positive proof that cross eye may be influenced by some diseased state of the nose.

In order to conserve time, I will not attempt to refer to the many reports relative to this point, but will relate, first, the history of a patient afflicted with alternating convergent strabismus, and then a case of alternating divergent strabismus. Both of these cases were cured by means of similar intranasal surgery, the results being fully as good as may be obtained by the usual operative procedures upon the eye muscles. Since attention to correcting refractive errors is a proven necessity in all treatments of strabismus, it is necessary for me only to add that this detail should never be neglected.

The first history is that of Mary M., aged 19 at time of first consultation, October 9, 1919. She sought relief for alternating convergent strabismus which had been under treatment by the use of correcting lenses since the age of five, or a period of fourteen years, without benefit. The relative facts of this interesting case are as follows: First consultation, October 9, 1919; last consultation, June 20, 1924; period under observation, 4 years and 7 months.

This patient had worn glasses since 5 years of age, to control high degree of compound hyperopic astigmia and relieve convergent strabismus which had been present since infancy. There had been no improvement of the strabismus by the recognized measures.

After the usual refractive tests, prescribed following:

$$\text{V. O. D.} = \frac{20}{70} \text{ plus 4 D. } \frown \text{ plus 3.50 axis 75} = \frac{20}{20}$$

$$\text{V. O. S.} = \frac{20}{70} \text{ plus 5 D. } \frown \text{ plus 1. axis 105} = \frac{20}{50}$$

Ophthalmoscopic examination; upper nasal quadrants of both optic nerveheads blurred. Condition of nose; thickened and spurred septum, cystic middle turbinated bodies and ethmoid. Corroborated by transillumination and radiograms. Teeth, good condition. Tonsils, fair size but healthy. No evidence of adenoids.

Advised intranasal surgery and later strabismus operation if required. The operation was performed May 5, 1920.

Full and extensive submucous resection, amputation anterior half right middle turbinated body. Bulla cells opened by single bite.

Postoperative Notes: Immediately following operation, urine showed trace of albumin against none before, and sp. grav. was 1032 against 1015 before.

Improvement in eyes speedily noted; general health improved. Convergent strabismus overcome both without and with correcting glasses. Photographs, June 1921.

This satisfactory result continued until left ethmoiditis ensued in February, 1923. Then the left eye slowly converged at times. Under intranasal treatment, this was readily overcome. It is interesting to note that negative galvanism, 4 m.a. current and voltage of 40, applied over vicinity of the left Meckel's ganglion, would cause a strong turning inward of the left eye.

Last report, condition fine and eyes turn only if overtired.

The second case is that of Margaret W., who first consulted me at the age of 10, September 24, 1913. This young girl presented the typical signs of alternating divergent strabismus and the essential facts in this instance are appended:

At age of 10 years, presented for first consultation because of poor vision. At age of 3 years, had had diphtheria, followed by scarlet fever and measles. Mother believed poor vision was a result of these diseases. At that time, eyes showed no signs of strabismus, although pupils were semidilated and exhibited sluggish reactions.

Physical examination negative. Urinalysis negative. Wassermann negative.

Ophthalmoscope showed hazy vitreous and marked papillitis in both eyes, retinal edema. Visual acuity and refraction:

O. D. $= \frac{20}{70}$ — 1.50 cyl. axis 180 $= \frac{20}{70}$

O. S. $= \frac{20}{70}$ — 1.50 cyl. axis 180 $= \frac{20}{70}$

Test under atropin:

O. D. $= \frac{20}{100}$ + .50 S. $\bigcirc$ — 1. cyl. axis 180 $= \frac{20}{70}$

O. S. $= \frac{20}{100}$ + .50 S. $\bigcirc$ — 1. cyl. axis 180 $= \frac{20}{70}$

Prescription: O. U. — 1. cyl. axis 180. Advised to discontinue school for one year.

Under simple treatment, but no iodides or mercury, and constant wearing of glasses, the eyes were improved, at the end of 12 months being:

O. D. $= \frac{20}{50}$ — 1. cyl. axis 180 $= \frac{20}{20}$

O. S. $= \frac{20}{50}$ — 1. cyl. axis 180 $= \frac{20}{40}$

At this time, the nasal half of the optic nerveheads were the socalled wooley type, but the temporal halves were fairly clear. Pupils were smaller and more active. Patient permitted to resume school.

February 15th, 1915. Refraction changed and improvement in vision retained. Glasses prescribed,

O. U. — 50 S., giving $\frac{20}{40}$ O. D. or O. S. or O. U.

During following two years, vision of O. S. was less acute than O. D. at the time of acute head colds (rhinosinusitis), and after mydriatic test, glasses again changed July 11, 1917:

O. D. — .25 S. ⌒ — 1. cyl. axis 180 = $\frac{20}{40}$

O. D. — .25 S. ⌒ — 1. cyl. axis 180 = $\frac{20}{40}$

At the age of nineteen, and six years after first consultation, the left eye showed a marked tendency to diverge, and vision with glasses O. D. = $\frac{20}{30}$, O. S. = $\frac{20}{50}$ so that at this time the right eye was becoming the fixing eye, and there was very definite papilledema of the left optic nervehead, and curiously, the right optic disc had become ischemic although visual acuity was unchanged.

By September 21, 1921, or eight years after first consultation, alternating divergent strabismus was positively shown, with right eye functionating to greater advantage.

V. O. D. = $\frac{20}{40}$; corrected = $\frac{20}{40}$

V. O. S. = $\frac{20}{200}$; corrected = $\frac{20}{50}$

O. U. Neuroretinitis, plus, plus.

During these periods, mouth breathing was becoming more and more pronounced and the nasal spaces more obstructed because of progressive cystic inflammation of the ethmoids and middle turbinated bodies, and by May 22, 1922, constant alternating divergent strabismus was evident, she being able to fix with either eye, at which time the fellow eye would rotate outward to the external canthus.

It is noteworthy, that under appropriate intranasal treatment and simple measures directed to the care of the eyes, the neuroretinitis of the left eye subsided and vision improved from $\frac{20}{200}$ to $\frac{20}{50}$. The vision of O. D. was unchanged, so that corrected vision at this time was O. D. = $\frac{20}{40}$, O. S. = $\frac{20}{40}$.

It is well to note that accommodative or ciliary spasm could be excluded, because the refractive state had already been determined under mydriatic before these marked symptoms became apparent, so there could be no reason for lessened visual acuity or involvement of the third nerve except some inflammatory state, which was believed to be secondary to the sinus involvement (ethmoiditis).

During these years, operation for removal of adenoids and tonsils was refused, and it was only because of the marked alternating divergent strabismus, which was a positive disfigurement, that consent was finally given to perform some intranasal operation. This was performed at the Memorial Hospital, May 23, 1922, approximately 9 years after the patient first came under observation. Opportunity had been thus afforded of witnessing the gradual development of an alternating divergent strabismus, which was coincident with progressive ethmoiditis and cystic enlargement of the middle turbinated bodies.

Papilledema of varying degree and changes in the visual acuity were

ever present until after relief was afforded by the intranasal surgery, these ocular symptoms being absolutely better or worse according to the varying pathology of the sinuses.

The operation was exploratory in nature, because the septum was irregular and all meati were blocked by the hypertrophic and cystic nature of the turbinated bodies. X-rays gave information as to the size of the sinuses and the fact that all cavities were clouded.

After extensive submucous resection, the flaccid membrane was pushed aside and the ethmoid regions inspected, after having first infracted the cystic turbinated bodies. This procedure permitted the free outpouring of a colloid mucopurulent secretion. Complete middle turbinectomies were performed and the ethmoid, both anterior and posterior groups, opened. The sphenoid cavities and the frontonasal ducts were probed. The sphenoid cavities showed bogginess of mucous membranes.

After operation, there ensued a complicating otitis media of left ear necessitating myringotomy. Recovery was without other incident.

Result of operation. Cosmetic cure of strabismus with transient fusion faculty. This result was obtained within two weeks after operation. Subsequent to the intranasal surgery, there was marked improvement of vision with coincident clearing of the ocular media, and at last examination the fundi were normal and the optic nerveheads had assumed a healthy pink tone. When discharged, uncorrected vision was:

O. D. $=\frac{20}{30}$, O. S. $=\frac{20}{40}$ and corrected vision was O. D. $=\frac{20}{20}$, O. S. $=\frac{20}{30}$

In closing, it is well to emphasize that in neither of these cases was syphilis demonstrated, and that throughout treatment, mercury and iodides were never prescribed, nor, indeed, was any other form of antiluetic treatment ever instituted.

Dr. Robert Scott Lamb, Washington, D. C.: This subject, it would seem, should interest all of us who deal with refractive cases. The question arises in one's mind as to the proportion of these nonrefractive asthenopic cases in one's practice. Unquestionably, many of them are overlooked because of the fact, that patients are refracted and are dismissed as having been properly refracted, and perhaps not being comfortable, seek attention elsewhere. It should be borne in mind that the question of history taking should be uppermost in the mind of the examiner in these cases. I believe the history will in many cases clear up a number of etiologic factors. Of course we have no statistics, but I have an impression, gathered from a large experience with many of these cases of nonrefractive asthenopia, that they are of functional disturbance origin, and that disturbance is caused by malnutrition, faulty metabolism, induced in many instances by a disordered endocrin system, and that through that, and through great drains upon the sympathetic nervous system, a socalled vagotonia, a disturbance of the vagus function, is responsible for changes in the circulation of the eye, producing at times an increased intraocular tension, and at other times a very much reduced intraocular tension. In either event, there is bound to be a painful dysfunction of the ciliary muscle. So I feel that it is rather important to stress the functional, vagotonic side of these nonrefractive asthenopias.

SOME ASPECTS OF PRIMARY CATARACT.

LIEUT.-COL. HENRY KIRKPATRICK.

LONDON, ENG.

My first words must express my thanks to you for the high honor you have paid me by inviting me to be your guest, and to address you on this occasion. I recognize in it a tribute to the work of Elliot, Herbert, Smith, and other surgeons in India, and to the Madras Government Ophthalmic Hospital.

It is difficult to find an aspect of primary cataract which is unfamiliar to an assembly of this kind; I therefore propose to approach the subject from the direction of Southern India, and to describe to you the gross features of the varieties of cataract most commonly observed in that country, with some of the conditions which may influence their occurrence. You will thus be enabled to judge in what respects this aspect of the disease may differ from your own.

Senile cataract is, as elsewhere, the commonest variety. Juvenile cataract occurs, I think, with greater frequency in Indians than in the white races; of 4,762 primary cataracts seen during three years, the statistics of which I have available, 61, or 1.2 per cent, were juvenile. Lamellar cataract is, on the other hand, comparatively rare; in the same period only 8 cases were met with.

An observer is struck by three things in connection with senile cataract in Southern India: (1) The numbers affected by the disease, (2) the comparatively early age at which it occurs, and (3) the large number of hypermature cataracts seen.

When estimating the comparative incidence of the disease, one must remember that the proportion of competent surgeons to the general population is very small, and that the surgeon in the East draws his cases from a very wide field; yet, after making full allowance for this fact, the disease certainly does appear to be more common there than in temperate climates. The number of sufferers from cataract met

with on the roads and in the villages is quite remarkable. Out of 52,994 persons who were admitted to the outpatient department of the Madras Eye Hospital during three years, 6,659, or 12.5 per cent, suffered from cataract. The disease seems to occur still more frequently in the dry and arid districts of the north of India.

The age of the average senile cataract patient in South India is at least ten years younger than that of the patient in Europe. The average Indian appears to mature and to retrogress at an earlier age than the European does.

The high proportion of hypermature cataracts is, of course, due to the postponement of operation through lack of opportunity or for other reasons. The surgeon is thus afforded an excellent chance to observe the changes produced in different types of cataractous lenses by hypermaturity. An analysis of 2,078 cataract operations shows that 18.7 per cent of the lenses were hypermature.

Two distinct types of hypermature senile cortical cataract may be observed: (1) The Morgagnian; (2) the shrunken.

The Morgagnian variety constitutes quite three-quarters of the hypermature cataracts met with. Its leading characteristics are well known to you. In its advanced form, small, dense, white spots are seen in the anterior layer of the lens capsule; these result from a hyalin degeneration of proliferating foci of the lining epithelium. The capsular bag is fully distended by the milky fluid derived from the altered cortex. The nucleus moves freely in the fluid, and gravitates to the most dependent part; it readily comes into view if a light tap is made upon the occiput while the patient bends his head forward between his knees. The anterior chamber is usually rather shallow, and the pupil is uniformly white, except for the specks of capsular degeneration. The nucleus is brown, and should the case be at all long standing, is shrunken, with a smooth, almost polished, surface; its size irresistibly suggests that absorption has occurred. The Morgagnian fluid becomes absorbed in course of time, but, with its disappearance, the capsular thickenings may extend and a membranous cataract develop; rarely a transparent area in the capsule remains above the level of the upper edge of the nucleus, and allows some useful vision to be regained. Cases are sometimes seen in which a leakage of the fluid, often due to an injury, has occurred and has set up an iritis; the irritating

character of the Morgagnian fluid is shown in this way. Sometimes the small shrunken nucleus spontaneously passes through the pupil into the anterior chamber and causes a glaucoma or an iritis; it may be difficult to distinguish this condition from one which has resulted from an attempt to couch the lens.

One recognizes a Morgagnian cataract during the earliest stages of its development (1) by its tendency to swell, as evidenced by the diminished depth of the anterior chamber, and (2) by the numerous narrow, bluish white sectors on its surface. These cataracts develop quickly, and, owing to the rapid swelling of the lens, may give rise to a secondary glaucoma. Extraction with capsulotomy is inadvisable before maturity is reached since the narrow, ribbon like sectors are often difficult to evacuate, and are likely to cause trouble by becoming lodged between the lips of the wound. It may be remarked that an attack of secondary glaucoma is often followed by a rapid absorption of the Morgagnian fluid. I know that some high authorities would consider that I have placed the sequence of events in the wrong order; I think, however, that, in most of the cases I have seen, the onset of high tension has preceded the absorption of fluid cortex. Glaucoma secondary to a swelling cataract is met with in 0.25 per cent of all patients admitted to the Hospital, *i.e.* about fifty cases of the disease are seen in a year.

The shrunken form of hypermature cataract is characterized by the dense and brittle nature of its degenerated cortex. During its development, absorption of fluid takes place from the cataractous matter, and inspissation occurs. This inspissation is specially marked in the equatorial region, and large, cheesy fragments of the upper portion of the lens readily break off during the delivery of the nucleus; these may remain concealed in the recesses of the capsule. In some cases the nucleus is flattened from before backwards, but ordinarily it is otherwise undiminished in size. Capsular thickenings usually occur as patches, and are often marked in the pupillary area. The depth of the anterior chamber is normal as a rule, but may be increased in advanced cases. A fine granulation can often be observed in the cortex exposed by the pupil; a faintly yellow tint is sometimes present also. The earliest stages of this type are difficult to recognize. My impression is that the early sectors are broad and thick, and

that they commonly originate in the less superficial layers of the cortex near the equator. Liquefaction of some narrow, superficial sectors may occur as the disease advances, but absorption of fluid and inspissation follow later. Progress is slow, but frequently this type of cataract can be safely removed before it is fully mature. Shrinking cataract constitutes nearly one quarter of the hypermature senile cataracts operated upon in Madras. It is possible that the condition may be a development of Vogt's "wreath shaped" cataract.

Owing to the comparatively early age at which both these forms of cataract occur among Indians, the cataractous cortical matter is usually more abundant than in Europeans. The lens nucleus is clouded, and is almost always darker than that commonly seen in Europe.

The nuclear form of senile cataract is much more rarely encountered than the above varieties. In it the nucleus is large and hard, and has a dark brown color; the cortex is usually small in quantity, and may show a few opaque sectors. Lenticular myopia is often an early symptom. Under focal illumination, occasional greyish sectors and striae may be noticed in the pupil against the dark reflex from the lens. The red reflex from the fundus is much diminished in intensity, but is seldom wholly lost. The cataract develops very slowly, but it may be extracted without undue risk whenever visual failure causes grave inconvenience.

A nuclear cataract may be met with at so early an age as forty; it is then unlikely to be very brown, but the lens may have a diffuse, faintly green reflex, which is rather suggestive of that sometimes seen in chronic glaucoma. Early extraction gives good results in such cases.

An analysis of 1613 operations for senile cataract shows the following percentages. Swelling type, 57.6; shrinking type, 33.9; nuclear, 8.5. I am indebted to Major Wright and to Messrs. Muthayya and Sridharan of the Madras Hospital for most kindly abstracting these particulars from my operation notes.

The juvenile cataracts seen are mostly of the swelling type which ultimately becomes Morgagnian. A well developed, firm nucleus may sometimes be present at a surprisingly early age.

The comparatively rare occurrence of both lamellar cataract and rickets in Southern India is noteworthy; keratomalacia occurs, however, with remarkable frequency.

Let us now glance briefly at the etiologic aspect of primary cataract.

I cannot but think it most important that we should consider the etiology of primary cataract as a whole, and avoid the separate discussion of each variety—such as senile, juvenile, lamellar and diabetic. All lenticular cataracts, except perhaps some of purely developmental origin, are simply opacities caused by a degeneration of the lens fibers.

The fibers of the lens are cells derived from the ectoderm. These cells are in various stages of growth and of senile change; the younger lie on and near the lens surface, and the older lie deeper, while the remnants of those formed in early fetal life are found at the center of the structure.

Cell degeneration results from disturbances in the quantity and in the quality of the nutriment. The effect of gross disturbances is seen in secondary cataracts. Primary cataract, on the other hand, follows less violent disturbances; the nutritive requirements of the lens fibers are very limited, and the response to less noxious influences is therefore slight and tardy.

Disturbances of lens nutrition, when sufficient and continuous, are likely to cause a progressive type of cataract; but, if such disturbances remain active for a limited period only, the consequent degeneration may be nonprogressive, and the opacity thus caused may become buried in the lens substance by the subsequent growth of healthy fibers around it. Cataracts of early life, such as those of the lamellar or of the punctate types, can be accounted for in this way. It is possible that, when a considerable portion of the lens fibers has degenerated, the process may continue, even though the primary causes have ceased to be active.

Many observations have been made upon the nature of the chemical changes which accompany or result from the degenerative process. Burdon-Cooper has noted the presence of tyrosin; and he attributes the formation of this amino-acid to a hydrolysis of the lens albumin. He suggests that the dark coloration, found in some cataracts, may be due to the oxidisation of tyrosin by a ferment which became active in the presence of light. Tyrosin occurred most abundantly in those cataracts which were associated with some degree of deficient renal function. Leucin, cholesterin, and other crystalline substances have been found in the cataractous lens. Burge ob-

served a very marked decrease in the potassium content, and a great increase in the calcium, magnesium, and sodium content of the ash obtained from cataractous lenses when compared with the normal structure.

Burge also found that ultraviolet light produced opacity in lenses suspended in a medium containing calcium and magnesium, but failed to do so in the absence of the salts. Calcium was observed to be most abundant in cortical cataracts, while sodium chlorid was in excess in the nuclear types.

Lenticular opacity may be experimentally produced in rabbits by the action of infrared rays (Vogt), and also by naphthalen poisoning. It has been observed frequently to follow experimental destruction of the parathyroid glands. Exposure to ultraviolet rays appears unlikely to produce opacity under ordinary circumstances.

Senility and diabetes are the conditions most commonly associated with cataract. The disease also occurs in connection with tetany and with acutely exhausting diseases, such as cholera. Lamellar cataract has frequently been observed in patients suffering from rickets. The presence of hypothyroidism has fairly often been noted in cataractous patients; but cataract does not appear to attack sufferers from goitre with exceptional frequency. I have long thought that much evidence exists which suggests that minor disorders of endocrinal function may play a very important part in the production of primary cataract.

Most surgeons now would consider that some constitutional and general change or defect is a most important element in the causation of primary cataract. Such a cause is suggested (1) by the bilateral character of the disease, (2) by the exacerbation which so often follows illness or a severe accident, and (3) by the fact that a hereditary predisposition to the disease is frequently present. It is likely, however, that a purely constitutional defect alone is seldom sufficient to produce an extensive degeneration of the lens, and that a further factor or factors, which act directly on the structure, are necessary. The precise part played by each probably varies considerably in different cases.

A very potent constitutional cause might be expected to act with greatest effect upon the growing fibers in the equatorial region, and upon those which lie beneath the anterior layer of the lens capsule, as occurs in diabetic cataract. Vogt's

observations upon the existence of different surfaces in the substance of the lens suggest that the fibers in the vicinity of one of such surfaces may sometimes be subjected to injurious strains during the act of accommodation, and may thus be rendered sensitive to such deleterious influences as are only slightly active, but are long continued.

The inhabitants of Southern India are subject to the following conditions which may predispose to cataract:

(1) Exposure to the heat and glare of a tropical sun.

(2) A diet which is often badly balanced and is commonly deficient in vitamins.

(3) Excessive inbreeding.

(4) A liability to gastrointestinal disease and to diabetes.

(5) A high rate of syphilitic morbidity.

Exposure to Heat and Glare.

The ability of infrared rays to produce a cataract has been experimentally proved by Vogt. The cataract found in furnace workers and in glass blowers affords further evidence regarding the harmful effect of light and heat under certain conditions. The roughly circular shape and the rather sharp outline of the opacity in this form of cataract suggest the influence of the pupil in limiting the number of the injurious rays; while its situation in the posterior cortex, close to the nuclear surface, suggests that the rays are reflected from the concavity of the posterior layer of the capsule onto a part of the lens where resistance is lowered by accommodative strain. Cataracts of this type are fairly often met with in Indians who have never been exposed to an artificial glare. The fact, that only a relatively small proportion of glass blowers and furnace workers contract the disease, indicates that some other factor is usually required to enable the rays to produce lens degeneration.

The evidence thus greatly supports the theory that glare, especially tropical glare, has a deleterious effect upon the health of the lens fibers. It must be remembered, however, that other tropical countries, in which equal glare is experienced, do not appear to show as high an incidence of cataract as India does.

Diet.

A lack of fresh vegetables is present in the diet of most Indians, and the consequent poverty in essential food

factors is likely to be enhanced by the probable paucity of vitamins in vegetables, etc., grown during the hot season. The frequent occurrence of beri-beri, of scorbutic conditions, and of keratomalacia is suggestive of this vitamin deficiency. Rice is the staple food in Southern India, and usually the carbohydrates in the diet are in considerable excess. McCarrison has found that a diet which is rich in starch and poor in vitamins leads to a disorded function of the whole endocrin system of the pigeon; this suggests that such a diet may injuriously affect endocrin efficiency in man.

Inbreeding.

Consanguineous marriages are extraordinarily common in Southern India; indeed, among many communities it is the custom for a man to marry his first cousin—the daughter of his mother's brother; among others, a marriage between a man and his niece—the daughter of his sister—is the usual one.

On taking a census of 434 male outpatients attending the Madras General Hospital, over 60 per cent stated that they were the product of such unions. A similar census of about 800 coolie emigrants, who came from all parts of the Madras Presidency, showed that 51 per cent were the offspring of such marriages. It is a common experience to be informed by a patient that his ancestors for the preceding four or five generations were first cousins. Such excessive inbreeding must seriously affect cell vitality and must predispose to degenerative changes. Any hereditary predisposition to cataract, too, is certain to be intensified.

Gastrointestinal Disease and Diabetes.

The intestinal flora and fauna of a resident in the tropics are numerous and varied. Inhabitants of the tropics who remain free from gastrointestinal troubles, ranging from a slight catarrh to severe amebic dysentery or cholera, are few and far between. It has been truly remarked, that East of Suez, the center of gravity of disease shifts from the thorax to the abdomen; a chill, likely to cause a cough or a cold in a temperate climate, is more often followed in the tropics by a colic or by diarrhea. Helminthiasis is extraordinarily common; and at least 70 per cent of the general population in Southern India suffer from hookworm disease (principally Necator americanus). Practically every well-to-do Indian of middle age

suffers from severe pyorrhea, or from some form of oral sepsis. It is unnecessary to discuss the harmful effect of oral sepsis upon gastrointestinal disease and vice versa.

Diabetes is very prevalent among the richer Indians, but is less commonly met with among the poorer classes; quite often there appears to be a causal relationship between pyorrhea and glycosuria.

Syphilitic Morbidity.

Syphilitic infection is remarkably frequent. This is demonstrated by clinical observation and by postmortem experience. Competent observers have estimated the rate to be so high as 25 per cent of the urban population, and from 5 to 10 per cent for the rural districts. It is noteworthy that, although syphilitic infection is so common, one rarely meets with the grosser manifestations of untreated syphilis, such as palate perforation, etc. Some slight degree of immunity to the disease appears to have been acquired.

Senile cataract seems to occur with much greater frequency in the Punjaub than elsewhere in India. Colonel Henry Smith has remarked that this greater incidence is limited to the plains, where wheat forms the staple article of diet; and that in the hills, where rice and barley are eaten instead, cataract is no more prevalent than it is in Europe. He has also noticed that stone, a disease more commonly found in the Punjaub than in any other part of the world, affects precisely the same areas as cataract does. This very interesting observation suggests that some factor or factors, commonly present in these districts, render the inhabitants liable to a disturbance of calcium metabolism, and that this derangement may have an important bearing on the etiology of cataract as well as of stone. The influence exercised upon calcium metabolism by the endocrin organs, especially by the parathyroid, is noteworthy in this connection.

Consideration of the above conditions, to which a population showing a high incidence of cataract is subject, shows that their general tendency is to lower cell vitality and to favor degenerative changes. Some, such as diet deficiencies, diabetes, and intestinal disorders, are often associated with endocrin disturbances. It is believed that endocrin irregularities can originate trophic disturbances in ectodermic tis-

sues other than the lens, and it is suggested that endocrin disorders may prejudice the health of that structure also.

Senility is the accepted cause for most primary cataracts; but one must feel that this is too vague, and that an endeavor should be made to determine the particular factor present in senescence, which is responsible for the degeneration of the lens cells. The fact that the majority of elderly people do not suffer from progressive cataract indicates either that such a factor is not present to an equal extent in every aged person, or that it becomes operative only in the presence of other complementary or supplementary causes. Senility is usually associated with a gravely defective action of the generative glands; this gonad deficiency must disturb the normal harmonious working of the endocrin system, and must call for a compensatory effort on the part of the other glands. Satisfactory compensation is likely to occur in most cases, but its establishment may sometimes be rendered difficult, or may be prevented, by the existence of other factors, such as microbic infections of the alimentary canal and elsewhere, food deficiencies, and hereditary influences.

Cataracts which, except in nuclear development, are in all respects similar to the senile types, occur fairly often in patients under 40 years of age. It appears rather unreasonable to attribute these to any purely senile change. Some systemic disturbance, which commonly accompanies senescence, may, however, be present in these patients; and experience shows that minor degrees of endocrin deficiency, insufficient to cause very obvious symptoms, may exist at any age. The effect of such deficiency is likely to become more apparent if other deleterious factors are associated.

Cataract has fairly often been observed to follow parathyroidectomy and tetany; and this also suggests that endocrin disturbances—especially of the parathyroid gland—may play an important part in the constitutional cause of the disease.

Both experimental and clinical experience indicate the importance of glare in the production of lens degeneration. Vogt has proved the harmful action of infrared rays upon the lens of the rabbit, and Burge's experiments hint that ultraviolet rays, under certain conditions, may be injurious to the living lens. The existence of any fluorescence in the lens tissue may render it less resistant to injury by rays of short wave length.

Most of us must have remarked the connection between cataract and errors of refraction. Ametropia and heterophoria, by causing changes in the local circulation, often appear to render the ocular tissues vulnerable to the effects of constitutional disease. Some portions of the lens cortex may undergo greater stress than others during accommodation, and may thus be rendered more sensitive to injurious influences. It must be remembered, too, that the tension of the zonular fibers during accommodation may, perhaps, sometimes produce areas of uneven capsular pressure upon the lens fibers.

Consideration of the etiology of cataract suggests that persons approaching middle age, whose family history shows that they inherit a liability to the disease, should be particularly careful to protect their eyes from glare, and should wear glasses which suitably correct any error of refraction and prevent any excessive accommodative strain. Senile change must eventually affect the tissues of all who live sufficiently long; but its onset may be postponed and its progress may be delayed by observing the ordinary rules of health. Organotherapeusis, too, may benefit those patients who show any sign of endocrin disturbance during the time of the climateric.

Each of us regards the operative aspect of primary cataract from his own particular standpoint. We are able to achieve success in a very high percentage of cases, and we are fairly well satisfied with our methods and with our results. I think, however, most of us would confess that, owing to various causes, a certain number of our patients fail to obtain the healthy eye and the high standard of vision we had hoped for; we therefore constantly endeavor to improve our technic and usually are prepared to incorporate in our methods any procedure of proved utility.

It is obvious, of course, that a technic suited to the needs of a particular patient and employed by a particular surgeon is not necessarily the best for another patient, especially if employed by another surgeon. Two principles, however, govern all cataract surgery: (1) The maintenance of the highest possible degree of asepsis, and (2) the removal of the maximum amount of lens matter consistent with the infliction of a minimum of trauma and of disturbance of the ocular tissues.

One regrets the fact that, owing to the delicacy of the membrane, conjunctival asepsis cannot be achieved by any rapid chemical method which destroys the bacteria in situ. Much can be done, however, by using eyedrops or applications containing silver nitrat, zinc, etc., during the weeks previous to operation.

A description of some features of Madras technic in the operation for senile cataract may, perhaps, interest you. The cortical matter is usually very abundant in the senile cataracts met with in Southern India, while many of the patients are not easy to control and do not readily carry out directions given them. These circumstances have led to the adoption of the following steps: (1) Irrigation; (2) preliminary capsulotomy; (3) a modified form of lens delivery; (4) the employment of lid control.

Herbert's method of conjunctival disinfection, by forcible irrigation of the fornices with perchlorid of mercury lotion shortly before operation, has been found of the greatest value in Madras. Degeneration of the superficial conjunctival epithelium is caused, and any microorganisms lodged among the surface cells are caught in a mass of mucus and catarrhal epithelium; this mass is sponged out of the fornices under a stream of saline solution. A marked reaction is present at the first dressing, but this disappears completely by the third day. Twelve eyes were lost from suppurative panophthalmitis in 5,092 consecutive cataract extractions performed during the years 1915 to 1919 at the Madras Hospital (0.25 per cent). If four of these are excluded, in which suppuration commenced later than the ninth day and was therefore unlikely to be caused by operative sepsis, the rate is reduced to 0.15 per cent. This record, however, takes no account of attacks of postoperative iritis, mild or severe, many of which should, of course, be attributed to infection at the time of operation.

Irrigation.

Irrigation was introduced by Colonel Elliot about the year 1900. Elliot, having seen McKeown employ the method in the treatment of immature cataract, at once recognized its value as a routine measure to remove the cortex and to perform the toilet of the eye in the operation for mature cataract. It is used for these purposes in practically every cataract extraction performed in the Madras Hospital; and the experi-

ence of many thousand operations amply proves that it does its work gently, safely, rapidly and efficiently. The shrunken nucleus of a Morgagnian catract, being too small to manipulate easily, may sometimes prove very difficult to deliver by means of pressure applied through the cornea. It can, however, be washed out of the chamber with ease by the stream from the irrigator.

Preliminary Capsulotomy.

Preliminary capsulotomy with a Bowman's needle was introduced by Drake-Brockman some 45 years ago, and has continued to be the hospital practice ever since. This proceeding has so many advantages that I find it difficult to understand why it has not been more universally adopted. The capsulotomy is effected immediately before the section is made. The surgeon, passing the needle into the chamber at the place where he proposes subsequently to enter the point of his knife, can see exactly what he is about, and can lacerate the anterior layer of the capsule with a precision difficult to equal by the employment of a cystitome, whose movements in an empty chamber can be seen only dimly through the collapsed cornea. Should the patient lose his self control and violently squeeze his lids while the section is being made, he is less likely to cause a vitreous escape if a capsulotomy has been performed, since the nucleus can be ejected from the capsular envelope leaving both the posterior layer of the capsule and the ligament intact. Another advantage of preliminary capsulotomy is that it enables us to estimate the size and nature of the cataract, and so to graduate the section made; it must constantly be remembered that, though too large a section may be disadvantageous, too small a one may be disastrous. Then again the ease with which the point of the knife enters the puncture made at the limbus by the needle is a further argument in favor of the procedure.

To reap the full benefit of preliminary capsulotomy it is necessary to dilate the pupil before operating.

Lens Delivery.

Lens delivery is effected by a maneuver which may be called "dipping." The first step is to press lightly with a curette upon the sclera behind the upper lip of the section. Gentle stroking movements are made in this region with the

instrument until the upper margin of the lens nucleus passes downwards through the capsular rent. As the lens slips downwards, the pillars of the coloboma spread out, or, if no iridectomy has been performed, the pupil becomes oval in shape. No further step is taken until this change is observed. The plane occupied by the ring of the limbus is next ascertained, and light pressure perpendicular to this plane is made with the curette upon the lower quarter of the cornea. It will be found occasionally that one pillar of the coloboma is more displaced than the other, or that the pupil, if intact, has formed an oval whose axis is not vertical. This indicates that the nucleus, when passing downwards, rotated slightly on its vertical axis, and that its anterior surface now looks somewhat towards the pillar which is undisplaced, or towards the side at which the lower pole of the distorted pupil lies. In such case, the pressure with the curette should not be applied over the vertical axis of the cornea, but slightly to the side of it on which the lower pole of the pupil, or the least displaced pillar of the coloboma, lies. If these steps are carried out correctly, only the lightest pressure on the cornea is required to cause the upper edge of the lens to appear between the lips of the wound. As the lens emerges, the direction of the pressure is altered to an upward one towards the section, and the lens is followed on its course.

This technic has been found to possess the following advantages:

(1) The lens can be delivered without using counterpressure.

(2) The nucleus never passes up beneath the scleral lip of the section. The danger that the lens may rotate on its transverse axis when pressure is applied is thus avoided.

(3) The cortical matter lying in the upper portion of the capsular envelope is dislodged when the nucleus is pushed down, and its evacuation is facilitated in consequence.

(4) The iris is subjected to a minimum amount of bruising during lens delivery. Since the nucleus is placed in a favorable position to slip through the pupil, or between the pillars of the coloboma, no pinching of the iris occurs between its edge and the margins of the wound. Risk of postoperative complications is thus greatly reduced.

(5) The delivery of the lens is easy, even though the patient persists in looking upwards. One prefers this posi-

tion of the eye, which is usually assumed by the patient spontaneously, as it entails less strain upon the hyaloid. One wishes to avoid frightening the patient by giving him instructions which he may find difficult to carry out, and should therefore be prepared to effect delivery regardless of the direction in which the eye is turned at the time.

In judging the direction in which to apply pressure when commencing to deliver the lens, it has been found advantageous to fix one's attention upon the ring formed by the limbus. The plane occupied by this ring is observed, and pressure is made perpendicular to it. The position of this plane is more easily recognized than that of the iris, and changes in it can be readily followed if the patient moves his eye. I have found that attention to this detail facilitates lens delivery from an eye turned up or in any other direction.

Smith's Method of Lid Control.

Smith's method of lid control is employed if the patient is restless and inclined to squeeze his lids together; or if, for any reason, there appears to be an exceptional risk of vitreous loss. The method is doubtless familiar to you all; and I am sure that those who use it will agree that it has almost as great a value in the operation with capsulotomy as it has when the lens is extracted in its capsule.

This maneuver (1) eliminates the action of the orbicularis, and (2) draws forward the orbital connective tissue with its attachments, and thus, by creating a negative pressure in the socket, sucks the posterior pole of the eyeball with the vitreous body backwards towards the apex of the orbit. The danger of vitreous escape is greatly diminished in consequence. To carry it out, the assistant stands on the patient's left side and passes a lid hook under the upper lid. The hook is held between the thumb and first finger of the right hand; and the three ulnar fingers of this are used to push the orbicularis muscle well above the superciliary ridge. In doing this the palmar surfaces of the fingers are turned towards the patient's feet. The lower portion of the orbicularis is held beneath the inferior orbital margin by the thumb or by two fingers of the left hand. Should a prolapse of the vitreous appear to be imminent, the assistant, still maintaining control of the orbicularis, pulls forward the lid upon the hook, and thus causes the vitreous to recede.

Smith's lid control needs a skilled assistant for its employment, and its application leads to a slight delay. The

stretching of the upper lid, too, is rather distressing to some patients. For these last two reasons it is ordinarily reserved for the kind of case already indicated.

The lid control usually employed is as follows: The assistant raises the speculum well off the globe with one hand, and with a finger or the thumb of the other hand he pulls the lower portion of the orbicularis downwards on the cheek. The surgeon, having completed the section, lifts the upper portion of the muscle above the superciliary ridge with the outer two or three fingers of his left hand, and maintains it in this position throughout the remainder of the operation.

Pressure of the upper portion of the orbicularis upon the levator palpebrae may be looked upon as the most important cause of a vitreous escape when the patient squeezes his lids together. Between its origin at the apex of the orbit and its insertion in the upper lid, the levator palpebrae muscle stretches along the upper surface of the globe like a band. If the insertion of the muscle is fixed by a speculum, any contraction of the orbicularis will cause pressure on this muscular band behind its attachment to the tarsus, and will tend to pull it forwards, so that it presses upon the posterior segment of the eye and is thus likely to squeeze out the vitreous. Danger of vitreous loss will be enhanced if the patient looks downwards while he squeezes; gaping of the section is then favored by the contraction of the inferior rectus, while the pressure of the levator upon the posterior segment of the globe is reinforced by the action of the superior oblique.

This control of the upper portion of the orbicularis and the maintenance of an upturned position of the patient's eye during lens delivery allow the surgeon to experience a great sense of safety. Control of the lower portion of the orbicularis by the assistant is less essential, but it affords increased security.

One thousand five hundred and eighty-six cataracts were operated upon in the Madras Hospital during the year 1919, and vitreous loss occurred in 34 of these 1586 operations—a rate of 2.14 per cent. Since many of the cases were complicated, and since those in which the lens had suffered the attention of a coucher are included, the rate cannot be considered high. I am convinced that it would have been far higher had we not employed the technic described. In a larger series of 4596 cataracts, vitreous loss occurred 108 times—a rate of 2.32 per cent.

THE MECHANISM OF PAPILLEDEMA.

WALTER R. PARKER, B. S., M. D.

DETROIT, MICHIGAN.

In general, there are three principal theories advanced to account for the causation of choked disc; namely, the inflammatory, the toxic or chemical, and the mechanical. In addition to these theories, it has been suggested that various combinations of the factors involved in them may be regarded as the possible cause of the swelling of the disc in cases of increased intracranial pressure.

The mechanical theory of the causation of choked disc depends on increased intracranial pressure, increased intravaginal pressure, disturbance of the return blood flow, and obstruction of lymph flow through the intravaginal to the subarachnoid spaces. Whether or not the increased intracranial pressure must be associated with increase in the amount of cerebrospinal fluid is still in dispute.

The original theory of von Graefe, that the increase of intracranial pressure caused by a tumor led to compression of the cavernous sinus, and that this gave rise to an engorgement in the ophthalmic vein, was disproved by Sessman, who showed that the ophthalmic vein had such free connection with the facial, that an obstruction to its flow into the sinus could not produce a choked disc.

The discovery of Schwalbe, that the intravaginal space at the optic nerve communicates with the subarachnoid space of the brain, led Schmidt-Rimpler to advance the theory, that when there was an increase of the intracranial pressure, a quantity of cerebrospinal fluid was pressed into the lymph space of the sheath of the optic nerve, leading to a papilledema and a secondary venous congestion.

Manz could not confirm the claim of Schmidt-Rimpler, that a system of canals in the lamina cribrosa communicated with the arachnoid space, but added to the Schmidt-Rimpler theory the suggestion that, by means of hydrops of the sheath, the intracranial pressure caused compression of the vessels

in the end of the optic nerve as they pass through the lamina, which pressure resulted in an edematous swelling and infiltration. This socalled Schmidt-Manz theory is accepted by many as offering the most satisfactory explanation of choked disc. Cushing and Bordley believe that the changes are due; first to mechanical, and second, to toxic or inflammatory causes. De Schweinitz says: "In general terms, it is probable that choked disc is produced by a combination of factors. In this combination, increased intracranial tension or pressure is most prominent, and the mechanical theory of its pathogenesis offers the most satisfactory explanation."

As the results of the experimental work which I have conducted seem to confirm the mechanical theory, I shall limit my remarks to this phase of the subject and not attempt a complete review of the literature.

In general, the intracranial pressure is exerted equally in all directions, and if this pressure be sufficiently increased, it manifests itself first along the paths of least resistance. The resistance at the disc to pressure from within would have as one element in it, the tension of the eyeball. The globe showing the lower tension would offer the lesser resistance, and the nerve in that globe would, therefore, be the one first to show an edema at the disc, if either nervehead were to become affected. Naturally, if the two globes had the same tension, both discs would be involved at the same time to an equal degree.

To test this theory, a series of experiments was conducted on dogs and monkeys after the following manner:

A sclerocorneal trephine operation was performed in one eye, in order to make the two eyes different as regards the tension. After a marked difference in tension had been established, the intracranial pressure was increased by various means that need not be enumerated at this time. It should be said, however, that in each instance the intracranial pressure was made on the side of the brain opposite the eye in which the tension had been reduced. The experiments were carried to a satisfactory conclusion in nine dogs and three monkeys. In every instance, the swelling of the disc appeared first in the eye showing the lesser tension. If the papilledema is not mechanical in origin, it is difficult to explain why the swelling of the nerve should always appear in the eye of lesser

tension—especially when the eye first involved was invariably on the side opposite the location of the tumor.*

While the results of these experiments were most convincing, confirmatory clinical evidence that the onset of choked disc is in the eye of lesser tension, is not always so satisfactory. The uncertainty of taking accurate readings of the tension of the globe, the lack of opportunity for making an early observation in brain tumor cases, together with the great difficulty of differentiating the inflammatory from the edematous changes, and the early from the late changes in the nervehead, all contribute to the complication of the problem. It is essential, also, to remember that a choked disc is only an incident in the life history of a brain tumor; that is, the disc becomes edematous, swells to a certain maximum degree, and in time the swelling recedes and atrophy may follow. Again, an eye that may show an initial lower tension may later, in the presence of an edema of the nerve with hemorrhages, show a secondary glaucoma that will result in a reversal of the relative hardness of the two eyes.

The difference in the tension of the two eyes is a determining influence in the onset of choked disc only in so far as it indicates which eye will be the first affected. The eye first affected need not necessarily be the one in which the disc shows the greater swelling at the time of observation. When both discs are affected, the first examination may reveal an early edematous change in one eye, while the other may show signs of beginning atrophy. The disc in the softer eye may show an older pressure with less degree of swelling than that seen in the nerve involved later.

In one instance in which the pressure of a brain tumor was suspected, and before any evidence of choked disc was present, I predicted the papilledema would appear first in the eye that tested the lesser tension of 3 mm. of Hg. Later, I had an opportunity of observing the swelling appear first in the nerve of this eye. Dr. James Bordley has had a similar experience.

The difference in the tension between the two eyes need not be very marked to be an influence upon the onset of papil-

*The original paper, entitled, "The Relation of Choked Disc to the Tension of the Eyeball," was published in the Journ. of the A. M. A., Oct. 7, 1916.

ledema, so long as the difference is constant. In the cases here reported, the minimum of difference as recorded on a Schiötz tonometer was 2 mm. Hg.

Why the maximum degree of swelling at the disc in cases of intracranial pressure is limted to seven or eight diopters is not certain. It may possibly be explained by assuming, that either the connective tissue in the lamina is put on the stretch with increasing resistance to the pressure from behind, or the edematous condition of the tissues at the site of the laminae acts as a stopper offering its own resistance. A combination of the two conditions might also be possible. Whatever the explanation may be, it is a well known clinical fact that the swelling of the disc never exceeds about 3 mm. in height.

The stretching of the tissue in the laminae in cases of posterior staphyloma, together with an atrophy of the disc, might offer a possible elucidation of the clinical observation, that choked disc is seen so rarely in patients with a high myopia.

A clinical study of choked disc in its relation to the tension of the eyeball was made in twenty-three proven cases of brain tumor. The cases studied in this connection may be classified in groups as follows:

GROUP I. Cases showing greater degree of swelling in the eye of lesser tension..... 6

GROUP II. Cases showing an equal amount of swelling of the disc with the tension equal in the two eyes. 10

GROUP III. Cases showing the greater amount of swelling in the eye of higher tension. Process older in the eye of lesser tension..... 2

GROUP IV. Cases showing the same amount of swelling in the two eyes but of different tension. Appearance of disc same in both eyes..... 5

In Group III, the process in the disc of the softer eye was distinctly older than in the other eye, although the amount of swelling was the same in each. This difference in the appearance of the two eyes would at least suggest the possibility that the swelling appeared first in the eye of lesser tension.

The tension in the cases in Group IV was different in the

two eyes, while the amount of swelling remained the same in amount and appearance, and these cases are, therefore, classed as exceptions to the rule.

Assembling the results as tabulated, eighteen cases or 78.2% conform to the rule, and five cases or 21.7% are exceptions or unexplained.

As a result of the experimental work and clinical observation, there were 100% of the cases in the experimental work, and 78% in the clinical, bearing out the theory that choked disc in cases of increased intracranial pressure manifests itself first in the eye of lesser tension, or in cases where the tension of the two eyes is the same, both become involved at the same time to an equal degree.

THE SIGNIFICANCE OF PAPILLEDEMA TO THE OPHTHALMOLOGIST.

JAMES BORDLEY, JR., M. D.

BALTIMORE, MD.

In 1908, Dr. Harvey Cushing and I published a series of papers outlining some clinical observations and experiments dealing with choked disc. In these papers, we expressed the view that the preponderance of evidence adduced by us strongly pointed to a confirmation of a mechanical causation. Time and additional evidence have but more firmly convinced me of the correctness of this idea.

Indeed, today I think of choked disc, or, if you will, of papilledema, only as a visible manifestation of an increase in intracranial pressure. The more firmly this view becomes fixed, the more certain do I become that the treatment of choked disc does not properly belong in the domain of the ophthalmologist. It is but a component of a symptom complex that clearly indicates a situation serious enough to demand immediate consultation. As choked disc is most frequently produced by intracranial tumor growth, the opthalmologist who delays consultation and full investigation assumes large responsibility. He is not justified in holding such a patient, even though the disc changes are slight and the vision normal. Vision is indeed a very unreliable basis upon which to predict the ultimate state of the nerve or even the duration of the choking. Choked discs may persist over long periods of time without in the slightest interfering either with the field or acuity of vision. On the other hand, without apparent alteration in the choking, the vision may undergo rapid and permanent deterioration. We can be sure of one thing: permanent alterations in the field and loss of acuity invariably take place if choked discs are allowed to run their course, and in a very large proportion of cases complete blindness will supervene before death terminates the story, and, indeed, only too often where the primary lesions will not lead to fatality.

Any permanent alteration in the true structure of the nerve will result in a corresponding change in sight, and it is well to bear in mind that this fact is not altered by treatment. Nor should we be unmindful that the longer the process continues, the greater will be the loss in vision; therefore, the ophthalmologist should lose no time in seeking expert advice outside of his own sphere of action. This advice should be sought no matter to what extent vision has been damaged because as Drs. de Schweinitz and Holloway have so aptly stated it: "All who have to do with the blind will realize how much it means to the sufferer if he has only light perception, and how he will cling to that small blessing with almost the same tenacity with which we should cling to the preservation of good sight. Therefore, it would seem, from even this standpoint, it is fair to give the patient a chance."

Choked discs are not always the result of tumor growth; they may prove to be the only manifestation of some hidden intracranial disturbance which spontaneously disappears, leaving behind no evidence of the causative factor. Undoubtedly, such cases have been confused with tumors, where symptoms other than papilledema were late in making their appearance. So I think it extremely wise, whenever we find a swollen nerve unassociated with other symptoms, to have tucked back in our minds that the intracranial tension resulting from some types of tumors is very variable, even at times permitting entire subsidence of swelling for periods of weeks and even months, and that choked discs frequently antedate other symptoms by months. Under the circumstances, it is unwise, I believe, to conclude that because a choked disc has diminished in size or disappeared entirely following some small extracranial operation, the focal infection prompting the operation must be the source of trouble. Experienced observers have all, I am sure, noted marked subsidence in swelling following operative shock and blood letting, only to see the choking back again in a few weeks.

In the past, attempts have been made to differentiate various types of choked disc. Unfortunately, there is nothing peculiar to a choked disc from any given cause to indicate that cause. We know only that when we find the papilla swollen, we have unearthed a symptom of some intracranial disturbance. To the physician looking for a solution of an immediate problem, this information is not very inspiring,

as it has called sharply to his attention a process which may lead to blindness and even death, without providing the means for an urgent diagnosis.

It is a comfort, however, to know that by a carefully executed process of elimination, we can generally arrive at a fair conclusion. We know, for instance, that swelling of the disc is extremely rare in simple meningitis, except where an obstruction hydrocephalus complicates the late stage of the meningitis, where it becomes almost inevitable. Its appearance is indeed a valuable indication of such a complication. Choked disc is likewise extremely rare in cerebral pyogenic abscess (doubtless because the brain is destroyed rather than pressed upon), except where there is considerable local edema, in which case there should be considerable rise in intracranial tension. This does not hold for tuberculous abscesses, which follow closely the symptomatology of brain tumor. In the choked disc of nephritis, we may have the typical picture of tumor or find it associated with macular figure, depending upon whether there is a simple cerebral edema or edema complicated by extensive vascular changes in the retina. Syphilis is probably the most frequent of all "medical causes" in the production of choked disc, and its presence should of course always be determined.

Foci of infection remote from the brain and optic nerve may be provocative of choked disc. Such a complication is, however, extremely unusual, and such a cause, while requiring investigation, should not be too seriously stressed in the mind of the investigator. As an instance of focal infection, I mention only in passing, nasal sinus disease. It has been my privilege to see nasal sinus disease complicated by choking of the discs, but in every instance where the cranial contents were viewed, before or after death, there existed unmistakable evidence of an intracranial complication. I have no idea what Dr. White may have in mind to say about this phase of the subject, nor do I wish to encroach upon his preserve, but I cannot discuss choked disc from the point of view of the ophthalmologist without pointing to the prevalence of the exaggerated importance attached to sinus disease as a cause of choked disc, and its possible serious consequences upon the individual of this mistaken notion. Let me illustrate: Recently, I had the opportunity of studying two patients, one in the service of Dr. S. J. Crowe, and the other

a patient operated upon by Dr. Walter Dandy, both at the Johns Hopkins Hospital. The first patient, a soldier, applied to Dr. Crowe for an operation on his right sphenoidal sinus, because he had been told it would restore his sight. The sinus had previously been operated upon and was still obviously diseased. Dr. Crowe attributed the choked discs to a probable cerebellar tumor, and an immediate operation confirmed this view. One year, however, had been wasted waiting for the hoped for result from the sinus operation, and during that time the patient had permanently lost his sight. The sinuses of Dr. Dandy's patient had also been extensively operated upon. She had a large removable cerebellar tumor, but was so devitalized by the months lost to the ravages of her malady, that she barely survived the operation. Possibly in very, very rare instances, sinus disease may lead to an increase in intracranial pressure through a nonpurulent increase in the fluids of the intradural spaces, such as we see occasionally in disease of the mastoid process, but such a possibility must constitute a very unusual exception, and should not so mislead the examiner as to make him overlook much more probable causes.

The greater my opportunities for study, the more convinced do I become that choked disc is, in the great preponderance of instances, dependent upon tumors of the brain. It has been my good fortune to enjoy a very exceptional opportunity in the observation of this condition, and I have so frequently seen tumors become manifest after months and even years had elapsed since the discovery of the nerve swelling, that I have grown very skeptical of snap diagnoses and remote possibilities.

Cushing and I were unable in our study of the eyegrounds to discover anything of value in determining the size or structure of a tumor. Small benign growths in some situations will, by a mechanical effect, increase the intracranial pressure more than large, malignant infiltrating ones placed elsewhere. Further, as de Schweinitz has emphasized, the stage of the retinal process is no indication of the duration of the cerebral lesion, nor can we predict in a case of tumor, in the absence of choked disc, when this is likely to occur. The whole question resolves itself into the study of the character of the intracranial pressure and its effect on the cerebrospinal circulation, regardless of the histology, the situation,

or the duration of the neoplasm. A tumor so situated as to produce an obstructive filling of the ventricles will cause a general increase in tension, often of high degree and of rapid onset. Under such circumstances, the choked discs which are practically equal on the two sides may rapidly appear. As Dr. Parker has pointed out, the intraocular pressure plays a part in determining the side which will show the first swelling. Having discussed with him his theory, and having enjoyed the opportunity of seeing some of his painstaking experiments, I am convinced of the correctness of his views.

As all choked discs from serious causes run practically the same course, except where interrupted by treatment or death, it might be well to try to establish some standard of change for the determination of prognosis and treatment. Marcus Gunn attempted this by dividing the progress of the swelling into five stages. His classification of the physical changes in the disc and the result on vision of treatment seem so well authenticated by other observers, that it would be folly to attempt a new arrangement. As he describes it: The first stage is that of increased redness and blurring of the superior and inferior nasal disc margins; the second, beginning filling of the cup, with blurring of the temporal margin and a tendency of the edema to spread into the retina; third, decided increase in edema and elevation of the nervehead, associated with the forming of striae between the disc and macula, and retinal hemorrhages; fourth, mounding and opaqueness of the disc, disc and retinal exudations, associated with an increase in size and number of hemorrhages; fifth, decided subsidence in vascularity and increasing pallor with or without sinking of its prominence, contraction of the retinal arteries with thickening of their perivascular lymph sheaths and spots of degeneration in the retina.

As the damage to the nerve and retina in the first three of these stages is but slight, the vision is usually good, and the prognosis as to sight is excellent if treatment of the primary lesion is successful. Where the primary cause can neither be located nor eradicated, subtemporal decompression should be resorted to during the first three stages, because the dangers attending the operation are relatively few as compard with its benefits.

When the fourth and fifth stages are reached, it is almost inevitable that some deterioration of sight will follow, either

with or without operation. Fortunately, however, decompression prevents blindness, and occasionally restores some degree of vision even in these late stages. Indeed, if but a vestige of vision remains, I am apt, other things being equal, to advise operation. The suggestion of the operation should at this time never carry with it a favorable prognosis, because, after the edema subsides, the vulnerable nerve fibers are gripped and compressed by the new tissue formation in the nerve, and its effects can never be calculated. Occasionally complete blindness follows close on the heels of the operation

Knowing the disadvantage as well as the advantage of decompressive operations, the ophthalmologist may feel great hesitancy in advising operation in unchanging choked discs showing only the primary stages. He must, however, remember that tumors may continue to grow and still show a fluctuating degree of neuroretinal edema, even to complete subsidence of the process; that there may be periods of temporary relief to an obstructive hydrocephalus through escape of fluid; that spontaneous hemorrhages into the substance of a vascular tumor may cause a sudden rise in intracranial tension with a resulting elevation in the height of the discs, which subside again as the blood is absorbed; cysts which possess a variable degree of tension may develop in degenerated areas of a tumor; and, finally, an advancing growth may lead to a local cerebral edema in the immediate neighborhood, with temporary increase in tension and a swelling of its disc, which lessens as the edema passes away. These variations in intracranial tension may greatly lengthen the life of the victim's sight, but if the minute infiltration of the nerve is sufficient to provoke degeneration and atrophy, there will be a permanent visual loss. This degree of infiltration may be long delayed and may take place gradually, or it may come with astounding suddenness. It is my opinion, be the cause what it may, that in these protracted, socalled mild choked discs surgical intervention should be advised, because continued swelling, even without progressive change, is a sure indication that intracranial tension is not falling, and without such fall it can reasonably be expected that vision will ultimately deteriorate. If I am correct in thinking that increased intracranial pressure is the cause of choked disc, and am secure in the knowledge that choked disc produces blindness, what other advice could I give?

I have no desire to seem radical, indeed my chief cause for worry in the past has been my conservatism. Still, when one stops to think that a large proportion of the individuals unoperated upon lose their sight, and that palliative decompressive operations offer no serious menace to sight and not much more to life, if performed by a capable surgeon, why should we hesitate? Drugs are essential in certain underlying causes, such for instance as syphilis, but if tried they should not be persisted in beyond the danger point of the choking. It is better to relieve the intracranial pressure first and then administer the drug. One method promises sight, the other possible blindness. Neurologists and surgeons may base their advice upon the presence or absence of other physical signs, but for the ophthalmologist the saving of vision must be the determining factor. It makes little difference to the patient whether we hold to the mechanical views of Schmidt-Rimpler or the toxic theory of von Leber, so long as we open for him a way to preserve his sight.

PAPILLEDEMA IN OTO-LARYNGOLOGY.

LEON E. WHITE, M. D.

BOSTON, MASS.

The presence of papilledema in oto-laryngology is an inconstant, imperfectly understood and often disputed entity. Not a little controversy has arisen, especially between the brain surgeons and the rhinologists, as to the possibility of its ever originating from infections in the accessory sinuses, teeth or tonsils. It is hoped that from this symposium a better understanding may arise, that its importance in the diagnosis of the various intracranial infections may be more correctly stated, and that a more satisfactory explanation may be given of its presence in one case and absence in similar ones.

In brain abscess, meningitis and lateral sinus infections, the otologists desire to make a correct diagnosis as early as possible, and experience has demonstrated that considerable help may be obtained by frequent examinations of the eyegrounds, in spite of the fact that any of these intracranial infections can occur with a normal nervehead. As Jobson well says: "Absence of ocular symptoms does not justify the exclusion of intracranial involvement, but their presence, when considered in conjunction with other symptoms, is a valuable guide to a timely operation."

The writer felt that an analysis of a large number of these cases should prove of value, so the following data has been taken from the records of the Massachusetts Charitable Eye and Ear Infirmary of the ophthalmoscopic findings in one hundred and seventy-six cases of brain abscess, meningitis and lateral sinus infections arising from the ear, nose and throat. The fundi examinations were by some one of the ophthalmologists, usually the surgeon on duty, and many thanks are due Doctors Quackenboss, Spalding, Derby, Lancaster, Verhoeff, Thompson and others for their painstaking reports. The terms used by the various examiners differ somewhat; for instance, one uses the term "optic neuritis" to describe what another designates as a "blurring of the disc

margins;" so that while an effort is made, in classifying the fundi changes, to adhere as closely as possible to the words of the examiners, it was necessary to collect these reports into certain groups for study and comparison. It is frequently difficult to draw the line of demarcation between these various groups as, for instance, the blurring of the disc margins and a "commencing" or "early" papillitis. "Clinically," Eagleton says, "it is seldom possible to differentiate between an optic neuritis and a papilledema. * * The nervehead manifestations of brain abscess usually are a combination of both. * * The causes giving rise to them are not thoroughly understood."

Classification of the Eye Findings.

1. Marked papilledema, i.e., 2 diopters or over, usually with hemorrhages.
2. Slight or early papilledema, i.e., under 2 diopters, yet with some elevation of the disc.
3. Blurring of the disc margins—often designated as optic neuritis.

The above classes all showed some optic nerve changes, while the following had no change in the nervehead.

4. Dilatation of the retinal veins, i.e., enough change to keep them out of normal but in the percentages they are classed as normal.
5. Normal.

Brain Abscess.

Mention of the fundus findings were found in fifty-six brain abscess cases from middle ear infection and two from nasal infections, of which nineteen were cerebellar, thirty-seven temporosphenoidal and two frontal.

In the nineteen cerebellar abscesses, the fundi were normal in six; slight engorgement of the veins in two; blurring of the edges of the disc in two; slight papilledema in five; marked papilledema in four.

That is, eight of the nineteen cerebellar cases (or 42%) were practically normal, while 58% showed optic nerve changes. This differs considerably from the findings of Friesner and Braun, who state that "optic nerve changes were present in sixteen of the eighty-six cases," i.e., 18.6% as contrasted with 58% in my series. They also state "the condition found is usually an optic neuritis, but occasionally there is a choked disc." In my cases, nine, or 50% of all the eye changes,

were classed as papilledema and but two, or 8%, as optic neuritis. In three of my cases the changes were more marked on the affected side, in one on the opposite side, and the impairment of vision was slight.

In the thirty-seven temporosphenoidal cases, the fundi were normal in fifteen; blurred edges in four; slight papilledema in five; marked papilledema in thirteen; i.e., 60% showed optic nerve change. The nerve changes were more marked on the affected side in seven cases, while in only one case was the swelling on the unaffected side the more pronounced.

My findings are quite similar to Dickie's, who states that "the condition of the eyegrounds was noted in thirty-four cases, and in twenty-six of them there was some change, and the condition was generally more marked on the affected side." He refers to Adson who "found choked discs in nine out of his twenty-six cases."

In my thirty-seven cases of temporosphenoidal abscesses, there were twenty-six fatalities, nine of which showed marked papilledema, and three blurring of disc margins. Nine were normal, i.e., 65% had optic nerve changes. Of the eleven cases which recovered, four had marked papilledema, one blurring of the disc margins and six were normal, i.e., 45% had some optic nerve disturbance. Thus, the nerve changes were somewhat more common in the fatal cases than in those which recovered.

Of the twelve fatalities from cerebellar abscess, one had marked papilledema, three slight or commencing, two blurring of the disc margins, and six normal; i.e., 50% had optic nerve changes.

Of the seven cases which recovered, three had marked papilledema, four blurring of disc margins; i.e., all the cases had some optic nerve changes, a rather interesting point when contrasted with the 50% in the fatal cases, and the 45% in the temperosphenoidal cases.

In the two frontal abscess cases, the fundi were normal although the spinal fluid was under pressure. This accords with Eagleton's findings: "In frontal lobe abscess, papilledema is rarely present. An examination of the records of over one hundred cases of frontal lobe abscess shows only three to have exhibited definite nervehead changes, all associated with symptoms of compression. The most uniform

absence of papilledema in frontal lobe abscess is due to the fact that papilledema is largely dependent upon an obstruction of the central cerebrospinal fluid circulatory system."

Meningitis.

Records of the fundi changes were found in sixty cases of meningitis from middle ear infection, and three from nasal infection.

The fundi were normal in the three nasal infection cases, although the spinal fluid in all was under pressure and contained bacteria.

Of the sixty meningitis cases from middle ear infection, three had normal fundi; two showed slight dilatation of retinal veins; sixteen blurring of disc margins; three slight or early papilledema; seven marked papilledema; i.e., 43% had optic nerve changes. Of these sixty cases there was a record of the lumbar puncture findings in thirty-eight. The spinal fluid was under increased pressure in thirty-two, while in six it was normal or subnormal. The fundi were negative in sixteen of these cases with spinal fluid under pressure, or 50%. It was normal in six and not noted in the remainder.

In the ten cases with papilledema, the spinal fluid was under pressure in seven, not noted in three. In the two cases with slight engorgement of the retinal veins, the spinal fluid was under pressure in all. In the sixteen cases with slight blurring of disc margins, the spinal fluid was under pressure in seven, normal in three and not noted in six.

In these sixty cases of meningitis, there were but two recoveries. In one, there was slight blurring of the disc margins, the spinal fluid was turbid, under pressure, 800 cells, but no bacteria were found. In the second case, the fundi were normal at first, later the "nasal borders were blurry, disc hyperemic, hylus obscured and fine vessels unusually distinct, veins distended and large vein in right showed pulsation," the condition being noted as one of early papilledema. The spinal fluid in this case was under pressure, turbid, 65,000 cells. Culture showed large cocci in short chains, probably steptococci. A later lumbar puncture showed spinal fluid clear, not under pressure, 270 cells, no growth.

Respiration became labored while removing the spinal fluid in one case of marked papilledema, and the patient passed out, probably from herniation of the brain into the foramen

magnum. It is always well to bear in mind this possibility when advising lumbar puncture.

The bacteriologic examination was recorded in twenty-eight cases. Two were staphylococci, fifteen were streptococci, five were pneumococci, six no bacteria were found. In the six cases with normal pressure of the spinal fluid, bacteria were noted in four, all streptococci.

In the thirty-two cases with spinal fluid under pressure, examinations for bacteria were recorded in fifteen. Streptococci were found in six, pneumococci in three, staphylococci in two, no bacteria in four.

In the ten cases with papilledema, bacteria were reported in three, all streptococci; no growth in two; no record in the remaining five. In the sixteen cases with blurring of the disc margins, the fluid was examined for bacteria in five; in four, streptococci were found, and in one no growth.

In the thirty-two normal fundi cases, the spinal fluid was examined for bacteria in fourteen; four showed streptococci, two staphylococci, five pneumococci, three no growth. The two cases with dilated retinal veins had streptococci in the spinal fluid.

It was interesting to note that streptococci were the only bacteria found in the cases with optic nerve disturbances, and that in none of the seven cases where other bacteria was found were there fundi changes.

The importance of early examinations of the fundi in suspected meningitis is well emphasized by Kopetsky, who states: "At an early stage the increase in the amount of cerebrospinal fluid produces a strangulation effect on the blood vessels of the optic nerve, and causes an eyeground picture which is fairly constant and highly significant. It results in a swelling of the papilla, and blurring of the outer margins of the disc, which must not be confounded with, nor is it the real choked disc seen later in the disease. This sign, taken in connection with what has gone before, is of great diagnostic value."

The sixteen cases with negative fundi but increased spinal fluid pressure is contrary to Beck's statement in discussing Dr. Jobson's paper on "The Ocular Symptoms of Brain Abscess and Sinus Thrombosis of Otitic Origin." He says: "The cause of optic neuritis, or choked disc, I think has been proved to be a mechanical impediment. Dail, of Prague, at

the Russian Medical Congress, showed the way the veins enter the optic nerve sheath. This fact led me to a study of the relation of the spinal fluid, or rather, the result we get from spinal puncture associated with choked disc. I have never seen a case of intracranial trouble in which the spinal puncture came with a marked spurt, that did not also show a choked disc. Conversely, in the absence of the choked disc the spurt of the spinal puncture has not occurred."

Lateral Sinus Infections.

Records of the fundi changes were found in sixty cases of infection in the lateral sinus. Of this number, a thrombus was found in thirty-six. The remaining twenty-four bled on incision of the sinus, although in some the vein walls were thickened and a mural thrombus was noted. Twenty of the thrombus cases had normal eyes, three slight engorgement of the retinal veins, five blurring of disc margins, two slight papilledema, six marked papilledema. Of the twenty cases without an obstructing thrombus, eight had normal eyes, one slight engorgement of the retinal veins, four blurring of disc margins, five slight or early papilledema, two marked papilledema.

40% of the sinus cases showed optic nerve disturbances. This differs considerably from Tobey, who states that "Optic neuritis or choked disc may be present in a small percentage of cases (10% or 11%) due to meningeal irritation and infection, or from extension of the thrombus and consequent blocking of the petrosals or even involvement of the cavernous sinus."

Lumbar puncture was done in eleven of these sixty cases. Fluid was cloudy in four, clear in seven, under pressure in nine, normal pressure in two. Of the nine cases of sinus thrombosis in which the spinal fluid was under pressure, fundi were normal in four, definite papillitis in three, slight blurring of disc margins in two. In the two cases with normal pressure of spinal fluid, fundi were normal.

Graham well states my views on the eye findings in lateral sinus infections when he says: "The stasis in the veins of the nervehead has been recognized for some years. It is so fleeting, and is so dependent in case of examination of the disc veins on the personal equation of the examiner, that one should be very careful about allowing the sign to weigh in

the diagnosis in doubtful cases. * * The headache, dizziness and choked disc are the most frequent of these signs, and have been more insisted on as diagnostic features on account of their comparatively early appearance. * * An explanation of the choked disc accompanying some cases showing the above symptoms is very hard to obtain, because it does not always occur, even in cases of thrombus involving the lateral sinus, the petrosals and the cavernous."

Artificial papilledema by compressing the jugular veins has always seemed to me a dubious method of diagnosis. Loeb refers to it as follows: "O. Beck and Crow found, that during compression of the internal jugular on the healthy side, a definite tortuosity of the central vein of the retina could be found on ophthalmoscopic examination, in jugular or sinus thrombosis. This fact is due to the interference with the venous return as a result of the compression of the healthy jugular, and to the presence of the obstruction in the sinus or jugular on the diseased side. The test is not always reliable."

Accessory Sinuses.

Of the cases of optic nerve involvement studied by me, about seventy-five in number, three have shown edema of the disc.

One (Case 5) was examined by Dr. Quackenboss, who reported the disc of one eye pushed forward about 2 D, considerable exudate on and about it, the edges lost, veins large and tortuous. Vision, fingers 1 foot. The other eye, while showing some blurring of the edges of the disc, had no swelling. Draining of the posterior sinuses was followed by a rapid disappearance of the edema of the disc, and vision of 20/20 was ultimately obtained, although there remained slight pallor of the nerve. This case was operated on in 1917, and had no further disturbance with the vision up to the time of his death from pulmonary tuberculosis, five years later.

The second case (Case 9) was referred by Dr. Verhoeff, whose examination showed marked unilateral engorgement and swelling of the disc of about 3 D, vision 20/200. This patient also had exophthalmos. There was involvement of the antrum and ethmoids. Vision rapidly returned after these sinuses were drained. This case has been seen at frequent intervals since 1918, and the vision has remained normal.

The third case, also unilateral, was referred from the Eye

Clinic of the Infirmary by Drs. Reimer and Verhoeff on May 12, 1924. They found edema of the right disc of about 2 D, vision nil. As I had this paper under preparation at that time, I was anxious to get all possible data. Several of the ophthalmologists confirmed the presence of edema of the disc. The loss of vision was of but four days' duration, and was preceded by an acute coryza. With the exception of a deviation of the septum to the affected side, with some blocking of the posterior sinuses, the nasal examination was negative. The radiograms of teeth, accessory sinuses and sella turcica were also negative. The optic canals were normal. The vision commenced to improve before a lumbar puncture or Wassermann could be taken, which was unfortunate as far as the study of the case was concerned, as the patient refused to return for these tests.

Rhinologic literature contains the reports of many cases diagnosed as papilledema, supposedly due to infection in the accessory sinuses, which have subsided after opening them. Surgeons dealing with brain tumors usually feel that there was an error in diagnosis, that the lesion was primarily in the brain, and that incidentally the condition subsided following, but not dependent on the intranasal surgery. Cushing says: "Only intracranial tension can produce choked discs; I am exceedingly doubtful whether inflammation of the sinuses can cause this symptom. A retrobulbar neuritis does not produce swelling of the nervehead. * * A choked disc is due to mechanical, not to inflammatory causes, and optic neuritis is a misnomer."

Undoubtedly there have been many cases with brain lesions and an accompanying impairment of vision, that have had the accessory sinuses exenterated not only without benefit, but also in some instances with serious or fatal results. Cushing justly criticises as follows: "But in any event, do not let us be misguided into the belief that no harm is done to patients with threatened loss of vision due to 'optic neuritis', if they have their turbinates removed and sinuses indiscriminately opened. No greater discredit could possibly come to rhinology as a specialty."

Thorough investigation would have prevented most of these useless procedures. But, on the other hand, the neural surgeons may overlook an accessory sinus infection and submit the patient to fruitless intracranial surgery. Meanwhile,

the vision may be seriously damaged, when it might have been possible to have preserved it by the elimination of infections in teeth, tonsils or sinuses. Sluder remarks apropos to this: "Neglect of rhinologic investigation in the presence of choked disc may also be basis for criticism, as a case referred to me eight years ago by Dr. W. H. Luedde shows. A normal woman, 30 years old, had had bilateral choked disc with severe headache. A bilateral brain decompression had been done, without betterment. Optic atrophy followed with loss of light perception. I found a bilateral sphenoiditis, and operated both sides. The headaches was relieved, and light perception returned. * * I had had the experience of turning the patient, losing vision, over to the neurologist for investigation, and before he finished his examination, which was elaborate but negative, the patient was blind beyond help, from sphenoiditis." Case 27 in my series became permanently blind while undergoing neurologic investigation for optic neuritis.

If, as Eagleton says, papilledema may result from some change in the composition of the cerebrospinal fluid, as happens from infection in a pneumatized petrous tip associated with an acute mastoiditis, it would seem within the range of possibility that infections in the posterior sinuses might likewise produce similar changes in the cerebrospinal fluid, to be followed by a papilledema. I have never seen such changes, which if they ever do occur, must be extremely rare.

This is as good an explanation as any I have seen, if it is a fact that the cases of papilledema reported by the following reliable rhinologists are identical with those produced by intracranial pressure from brain lesions.

Loeb reports two cases. One, with bilateral loss of vision, had a swelling of 4 D, in one eye and 6 D, in the other. The second case had a unilateral swelling of 3 D, with vision of light perception.

Wood and Wallis report a case with defective sight for two years in the left eye, vision of hand movements, and the right eye was also somewhat affected. Vision 6/9, with marked papilledema. There was pus in the nose. The removal of the middle turbinates and opening of the sphenoid brought about normal vision right, and nearly normal left.

Sluder mentions a case with a swelling of the disc of 6 D, in one eye and 8 D, in the other, arising from double sphenoiditis. He further states: "I have had like cases referred

to me by Drs. Joseph W. Charles, A. E. Ewing, John Green, Jr., William H. Luedde, W. A. Schoemaker and Meyer Wiener, all most competent ophthalmologists, in which the neurologic examinations were made by most competent neurologists. There was no instance, however, of so great swelling as 8 D. Many of these were in observation until complete recovery of the eye. * * Ophthalmoscope showed: right, mushroom like swelling of disc with swelling starting abruptly at margin and mounting up. Margins very much veiled, veins a little dilated and tortuous."

He continues: "Many times I have had eye cases referred to me with the statement of the most competent ophthalmologists, that there is a lesion, usually termed choked disc, of 1 to 8 diopters, and no neurologic or other cause can be found for it. I have been able to recognize hyperplastic sphenoiditis, with often acute processes added, in these cases, and have relieved the eye trouble by the postethmoidal sphenoidal operation."

Stucky reports "three cases of 'choked disc' due to suppuration in the sphenoid sinus or posterior ethmoid cells. * * Nearly all of them were complicated by a systemic condition. * * The diagnosis of the accessory sinus condition was verified by subsequent operation."

Stark, in a review of the literature in 1915, found five cases with choked disc among fifty-two cases of nerve involvement from the accessory sinuses. Of these cases, "one was from maxillary sinus, ethmoid and sphenoid trouble. One was posterior ethmoid. In the other three the location is not given."

Stark reports one of his cases: "A man, aged 34, apparently healthy in every way, reported to me that the vision of his right eye had materially decreased in the last few days. Examination disclosed central scotoma for color, pupil slightly larger than normal, no double vision, disc elevated 5 D, with the usual venous stasis, otherwise, it was normal. * * A Wassermann test was made which gave a reaction. The case cleared up in a short time under antisyphilitic treatment, leaving normal vision. To my mind, this must have been a case of syphilitic involvement of one of the posterior sinuses; otherwise we could not account for the improvement with suction."

From these reports by men of unquestionable skill and

integrity, reports which might be duplicated many times, the fact seems well established that there are cases of edema of the optic disc which arise from accessory sinus infections.

The next, and in a way the crucial, point is the one of nomenclature, and it is this nomenclature that is, I believe, responsible for most of the controversy. Are these edemas from the accessory sinuses identical with those from intracranial pressure? While both have swollen discs and appear alike, would it not be possible to differentiate them? In the edemas from intracranial pressure, the loss of vision is gradual or it may even be normal, while in those from the accessory sinuses it is sudden and usually complete. In the former there are no sensory disturbances about the eye, while in the accessory sinus edemas there is usually lameness and sensitiveness. In the edemas from intracranial pressure, there are generally other neurologic symptoms, while in those from the accessory sinuses there are usually none. It seems probable that in the accessory sinus edemas there are inflammatory swellings with resulting exudates in and about the nerve, which produce a much more serious constriction than those from infiltration of the cerebrospinal fluid. In many of the papilledemas associated with intracranial infections, we have the history of a suppurative middle ear, the patient is critically ill, his system is being overwhelmed by the septicemia and the eye condition is only an incident; while in the edemas from infections in the sinuses, the loss of vision is the distressing symptom, the infection is a localized one in and about the optic canal, and the general system is but slightly disturbed. In spite of the ophthalmoscopic similarity in the appearances of the nerveheads in these dissimilar conditions, there is little queston that etiologically we are dealing with two very distinct optic nerve disturbances. It would, therefore, seem infinitely wiser if we could designate them by different names. For instance, the swelling of the nervehead (which, let me interpolate, is very rare) from accessory sinuses, teeth or tonsils, being in the nature of a neuritis plus some swelling, would be well designated by the word "optic neuritis with edema", while the word "papilledema" should be reserved for the swellings associated with intracranial infections and neoplasms accompanied with increased intracranial pressure.

The following quotations from Sluder supports these views: "Neurologists, for the most part, think that choked

disc can be produced only by intracranial pressure, and that it is edema of the disc produced by cerebrospinal fluid pressed out in the sheath of the optic nerve. Ophthalmologists recognize what is the same lesion, or one identical with it, and indistinguishable from it, that certainly arises from other causes also. Neurologists sometimes think that they are the better judges of what is choked disc, which may account for some of this difference of opinion. * * * I wish to emphasize that choked disc is a lesion that seems to be of more than one etiologic mechanism; that the term choked disc describes a swelling of the nervehead and is not specific for its etiology; that the lesion secondary to postethmoidal-sphenoidal lesions is in all probability an optic neuritis; and that by the ophthalmoscope it may be indistinguishable from that produced by intracranial lesions."

There is one phase of this subject that needs special emphasis, i.e., loss of vision from the intracranial neoplasms in patients with definite nasal pathology. The ophthalmologic examination reveals a fundus that might be due either to early intracranial pressure or to accessory sinus infection, and the patient is referred to the rhinologist, who finds infected sinuses or suspicious tonsils. As this might be the explanation of the eye condition, there is likely to be no further attempt to investigate the case. The removal of the infection within the nose or throat naturally does not benefit the vision of patients with multiple sclerosis or acoustic neuromas, and it is in this way discredit comes to the science of rhinology. Cushing bitterly chastises us for neglecting to properly investigate these cases. Thanks to his prodding, they are now being studied much more thoroughly. It has been my good fortune in several instances to discover the more serious intracranial lesion and advise against nasal operations. The history of the onset of the amblyopia is so typical, either of its nasal or central origin, that this in itself should put us on guard. The slow progressive loss of vision, nearly always bilateral in the cases of central origin, warrants at least the necessary time for investigation, while in the sudden unilateral loss of vision cases, especially with normal optic canals, needless haste is unnecessary. Many of these will recover spontaneously. My three cases with swollen discs were unilateral, and all had sudden loss of vision. If the rhinologist would restrict his surgery to these acute unilateral cases, he would rarely have occasion

to be censured for unnecessarily opening the accessory sinuses. While bilateral edemas of the disc from intranasal infections have been reported, they are rare and need much more careful investigation, especially if the loss of vision is gradual.

My experience within the past three years has convinced me of the advisability of letting the accessory sinuses alone unless they show evidence of infection, and to look elsewhere for the focus. My only reservation would be that in those cases with abnormally small canals (4 mm or less), it might be necessary to ventilate the posterior sinuses as a therapeutic measure, to prevent optic atrophy. These are, however, very unusual cases.

Summary.

Papilledema when present in meningitis, brain abscess and lateral sinus infection is a valuable diagnostic aid, but its absence does not exclude intracranial involvement.

The data from the Massachusetts Charitable Eye and Ear Infirmary seems of especial value, as the eye examinations were made by ophthalmologists who were not particularly interested in the findings, i.e., their attitude was always neutral.

Nervehead changes are classified under three headings:

1. Marked papilledema.
2. Slight or early papilledema.
3. Blurry disc margins.

Dilatation of retinal veins being more or less of a transitory disturbance is classed with the normals.

Optic nerve changes were found in 58% of the cerebellar abscess cases and in 60% of the temprosphenoidal cases.

The fundi were normal both in the frontal abscess and meningitis cases arising from nasal infections. 43% of the meningitis cases from middle ear infection showed optic nerve changes.

Normal fundi were found in 50% of the meningitis cases with increased spinal fluid pressure. Streptococci were the only bacteria found in the cases showing optic nerve changes.

40% of the infected lateral sinus cases had optic nerve changes.

The fundi were normal in 45% of the lateral sinus cases with spinal fluid under increased pressure.

Edema of the disc was found in but 4% of the optic nerve disturbances arising from infections in teeth, tonsils or accessory sinuses.

Swollen discs from accessory sinuses, teeth or tonsils is an unusual condition, is transitory and is always associated with sudden and marked impairment of vision. Papilledema is the usual condition in brain tumors, and before advocating exenteration of the accessory sinuses, a very careful study of the neurologic side of the case should be made. In these edemas of the disc, the burden of proof is on the rhinologist, and I should advocate no intranasal work until the possibility of brain lesion has been eliminated.

The socalled papilledema from infections in the accessory sinuses, teeth or tonsils is not identical with that associated with brain tumors, brain abscess, meningitis and lateral sinus infections. It would seem advisable to designate the edemas associated with sinus infections as "optic neuritis with edema" and restrict the word "papilledema" to the edemas of the disc caused by increased intracranial pressure.

BIBLIOGRAPHY.

Jobson, J. B. Ocular Symptoms of Brain Abscess and Sinus Thrombosis of Otitic Origin. Trans. Amer. Acad. Ophth. and Oto-Laryng., 1914.

Eagleton, Wells P. Brain Abscess.

Friesner and Braun. Cerebellar Abscess.

Dickie, J. K. M. A Contribution to the Study of Brain Abscess. Trans. Amer. L. R. and O. Soc., 1922.

Kopetzky, S. J. Contribution toward the Early Diagnosis of Meningitis. Trans. Amer. Acad. Ophthal. and Oto-Laryng., 1912.

Tobey, G. L., Jr. Lateral Sinus Infection, Diagnosis, Treatment and Complications. Trans. Amer. L. R. and O. Soc., 1921.

Graham, H. B. Difficulties in the Diagnosis of Sinus Thrombosis. Trans. Amer. Med. Assn., Section L. R. and O., 1915.

Loeb, H. W. Operative Surgery of the Nose, Throat and Ear, vol. II.

Cushing, Harvey. Remarks on the Acoustic Neuromas and on Ethmoidal Operations for Choked Disc. Trans. Amer. L. R. and O. Soc., 1920.

Sluder, G. Borderland of Rhinology, Neurology and Ophthalmology. Trans. Amer. L. R. and O. Soc., 1921.

Loeb, H. W. Two Cases of Blindness Relieved by Ethmoid Exenteration. Trans. Amer. L. R. and O. Soc., 1920.

Wood and Wallis. A Case of Bilateral Papilledema and Ring Scotoma Due to Sphenoidal Sinusitis. The Lancet, 1910, p. 937.

Stucky, J. A. Some Serious Eye Conditions the Result of Intranasal and Nasal Accessory Sinus Disease. Trans. Amer. Acad. Ophthal. and Oto-Laryng., 1913.

Stark, H. H. Sudden Blindness due to Suppuration of the Accessory Nasal Sinuses. Trans. A. M. A., Section Ophthal., 1915.

Retrobulbar Neuritis, Secondary to Diseases of the Nasal Sinuses. Trans. Amer. L. R. and O. Soc., 1921.

White, L. E. Loss of Sight from Retrobulbar Neuritis due to Posterior Accessory Sinus Disease with Report of 17 Cases. Trans. Amer. L. R. and O. Soc., 1919.

Accessory Sinus Blindness, Differential Diagnosis and Operative Technic. The Laryngoscope, vol. XXXI, No. 8, Aug., 1921.

THE SIGNIFICANCE OF PAPILLEDEMA TO THE NEUROLOGIC SURGEON.*

GILBERT HORRAX, M.D.

BOSTON, MASS.

In attempting to arrive at proper conclusions concerning the significance of papilledema to the neurologic surgeon, I have used the avenues of approach which were at my disposal, with the object in mind of answering the following questions: First, what conditions other than brain tumor or its surgical equivalent can cause choked discs? Second, what has been the previously assumed etiology of the known papilledema in a considerable series of verified brain tumors, together with the various forms of treatment employed for relief? Third, what harm, if any, to the patient has resulted from the delay incident to treatment directed upon an erroneous assumption? To answer the first question, a study was made of all cases which have been registered on the surgical service at the Peter Bent Brigham Hospital with the diagnosis of a condition which either actually did, or conceivably might cause choking of the discs. For questions two and three, a series consisting of the last one hundred cases of verified brain tumors showing papilledema was investigated. These cases were in no way selected, and merely represented the most recently admitted patients having the conditions indicated.

Concerning the first problem, it was found that during the past ten years there were 183 patients other than those with brain tumor (including abscess, gumma and tuberculoma), whose diagnosis upon discharge represented a condition which we know may cause papilledema. Of these patients, however, there were 44 who had conditions which we may term "tumor equivalents." By this is meant conditions simulating brain tumor, for which an intracranial operation is indicated just as positively as in tumor. The great majority of these, namely 33, were patients suffering from chronic or subacute cisternal

*From the surgical clinic of Dr. Harvey Cushing, Peter Bent Brigham Hospital, Boston.

arachnoiditis. This condition has been called by different names, such as serous or cystic meningitis. Its etiology is uncertain, but may possibly be a cerebral infection allied to poliomyelitis. At all events, it produces a thickening of the arachnoid membrane, with a consequent backing up of cerebrospinal fluid in the large arachnoidal cisternae, especially along the base and around the cerebellum. This fluid accumulation simulates tumor so exactly, that it cannot be differentiated with certainty except by an exploratory operation. Such an operation, we believe is always indicated, because the condition is quite curable by this means, and although in some instances the choked discs might subside spontaneously, nevertheless they do not do so in all cases and the patient's vision should not be subjected to that hazard. The other two conditions of "tumor equivalents" were oxycephaly (8 cases), and pachymeningitis hemorrhagica interna (3 cases). The former all had a low grade of papilledema with slow, gradual formation of new tissue, and it is our belief that they should be decompressed in order to relieve the slight but constant intracranial tension, and thus preserve the optic nerves from further damage. The three patients with pachymeningitis also had choked discs, and were all cured by a complete mechanical removal of the organizing blood clot. These 44, therefore, should be aligned with the tumor cases, leaving a balance of 139 patients in the group other than tumor or its equivalent. These 139 cases represented seven conditions divided into the numbers shown in Table 1.

TABLE 1.

Diagnoses	Number of Cases	Number with papilledema
Sinusitis (all kinds)	52	1
Arteriosclerosis	36	4
Encephalitis	25	1
Tuberculous meningitis	9	3
Meningoencephalitis	9	0
Intracranial aneurysm	7	3
Cerebral thrombosis	1	1

Of the total number, there were 13 which were said to have shown choking of the discs, this being 9.4 per cent. Therefore, it will be seen at once that an extremely small percentage of conditions other than tumor had actually caused papilledema in the series at the Peter Bent Brigham Hospital. When this figure is compared with the percentage of tumor

cases causing choked discs, a figure given by different authorities almost universally above 85%, the discrepancy becomes evident.

This, then, is the first point, that in comparison to brain tumors or their surgical equivalents, other conditions causing papilledema are exceedingly infrequent.*

For the second portion of this investigation, the last 100 cases of verified brain tumors showing papilledema were used. Of these, it was found that 48 had been treated in one or more ways for their choked discs before being referred to a neurologic surgeon, while the remaining 52 were referred to this clinic as soon as the papilledema was discovered. In the group of treated patients, the most frequent procedure was a change of glasses, which occurred fourteen times. This form of therapy was no doubt instituted in most instances by an optician, but in not a few cases an ophthalmologist had been consulted. Other frequent procedures were the removal of tonsils and adenoids or the administration of some glandular extract. A complete view of the wide range of treatment, with the figures for each type, may be seen in Table II.

TABLE II.

Types of treatment carried out for alleviation of papilledema.	Number of instances of each type.
1. Change of glasses	14
2. Pituitary and thyroid extract	5
3. Tonsils and adenoids taken out	5
4. Gastrointestinal measures	5
5. Operations on nose and nasal sinuses	4
6. Symptomatic measures for "epilepsy"	4
7. Treatment for "neurosis"	4
8. Osteopathic treatment	3
9. Antiluetic treatment	3
10. Teeth removed	2
11. Miscellaneous (one each)	9
Total procedures	58

*It may be said that one other condition, namely, nephritis, may induce changes in the fundus that simulate choked discs very closely. It was impossible to investigate the numerous renal cases at this time, so that this group has been omitted from consideration. Urinary examination being such a routine, however, it must be very seldom that a differential diagnosis is not made.

To illustrate more in detail what has been the course of treatment in individual patients whose choked discs were not at once assumed to be due, in all probability, to a brain tumor, a few specific examples may be given. The cases chosen as illustrations represent both sexes, and ages varying from 17 to 48. Tumors of many kinds and in various situations are represented, such as gliomas, endotheliomas, and acoustic neuromas, so that it is obvious that no single tumor type is entirely responsible for the confusion, although in reviewing the 100 tumor cases, it appears that the acoustic growths are on the whole more likely to have had previous treatment on an erroneous basis.

The first case to be given was an example of the last named variety of neoplasm. This patient had consulted seventeen physicians before the cause of her varied discomforts was discovered.

Case 1. P.B.B.H. Surg. No. 14294. Left acoustic neuroma with advanced pressure symptoms. Visions reduced to light perception with the right eye, and to 20/100 with the left.

April 4, 1921. Admission of Harriet R. K., age 26. She had first noticed impairiment of hearing in the left ear in July 1918, and consulted an otologist who believed that her difficulty was due to wax in the external meatus. The wax was removed without improvement in hearing. She returned to the same man 2 or 3 months later, and this time he thought that the condition was "catarrhal". The Eustachian tube was inflated, and she was told that the other ear could be saved from impairment. Some weeks later, the same procedure was carried out by a second aurist. No relief ensued. In December, 1918, after an attack of headache and vomiting, glasses were prescribed for hyperopia. She was also given a diet at this time, on the basis that her vomiting had been due to a gastrointestinal disturbance. In March, 1919, she was again examined, this time by an internist, and after a gastric analysis, rest was prescribed. During August, 1919, she began having severe headaches, which finally became localized to the suboccipital region. Gastrointestinal X-rays were taken, and she was treated with belladonna without relief.

In December, 1919, she first complained of blurring vision, and in January, 1920, her eyes were again refracted. She was treated for a gastric disorder for the third time during January to March, 1920. In the Summer of this same year, she noticed

for the first time some staggering and unsteadiness. These symptoms progressed rapidly, and by January, 1921, her gait had become "drunken" in character. By this time, also, her vision had failed to an alarming degree, and after consulting an "oculist", she was told that she was losing her sight because of some focal infection. Her appendix consequently was removed in February, 1921, while her tonsils and adenoids were excised a month later.

Upon admission to the Brigham, she presented the full-blown syndrome of a left cerebellopontile angle tumor. Vision O.D. showed light perception only, while V.O.S. was reduced to 20/100. Her fundi showed receding papilledema, with advanced secondary atrophy consequent to it.

On April 8, 1921, Dr. Cushing exposed and partially removed a left acoustic neuroma. She was discharged to her home May 3, 1921. Her pressure symptoms were relieved and her ataxia improved, but her optic nerves had been damaged beyond the point of very useful vision before her intracranial operation could be undertaken.

COMMENT: The foregoing case was perhaps an extreme example, especially among the more recent patients, but such a story of delay was not infrequent among the earlier records of even five to ten years ago. There was no information in the history as to the earliest appearance of the papilledema in this patient. Her vision, however, began to fail in December, 1919, and although choked discs may be present for a long time before visual impairment is noted, the reverse could hardly occur. In addition, her fundus examination at the Brigham showed that her optic nerves had long since passed through the more acute choked stage, and had gone on to nearly complete secondary atrophy.

The next case was likewise an angle tumor. The record of this patient gave a definite statement concerning the onset of papilledema.

CASE II. P.B.B.H. Surg. No. 12722. Left acoustic neuroma with extreme impairment of vision, marked cerebellar ataxia and general pressure symptoms.

July 3, 1920. Admission of John C. R., age 25. Illness began in Sept., 1919, with headaches, which were soon followed by vomiting and dizziness. On Oct. 14, 1919, he consulted an ophthalmologist, who discovered that he had what was termed "optic neuritis". He stopped work in March,

1920, and consulted another ophthalmologist, who noted that he had "double choked discs, easily +4 D, on each side", and also noted that there were "nystagmic jerkings" to right and left. This second ophthalmologist thought of the possibility of brain tumor, but influenced it appears by a neurologic colleague, attempted to explain the fundus picture on another basis. In discussing this question he said, "It is difficult to classify these 'choked discs' with the socalled spurious optic neuritis of advanced degree. To be sure, the appearances of the choking are not exactly those seen in the papilledema of brain tumor, but even so they seem to be greater than those of spurious neuritis."

The patient's vision continued to fail gradually until June, 1920, after which there was rapid impairment until he was admitted to the Brigham Hospital.

His examination upon entrance showed marked ataxia and incoordination, with positive Romberg's sign, persistent nystagmus to right and left, and some tenderness to pressure in the suboccipital region. It is remarkable, in view of the subsequent tumor verification, that he showed no loss of hearing in either ear at this time, either subjectively or by the usual tests. The eyegrounds showed a bilateral papilledema of 4 D, along with advanced atrophic changes and much new tissue formation. There was complete embedding of the vessels in many places. Vision was reduced to 20/70 with the right eye and 7/200 with the left.

On July 8, 1920, the cerebellar region was explored by the writer and no tumor disclosed, although intracranial tension was great. The pontile angles were not explored, and the operation left as a decompression.

This patient improved subjectively in everything except his eyesight, showing that although his pressure had been removed, his optic nerves had already suffered beyond repair. He went along very well for three years, when his ataxic symptoms again became more marked and he returned to the Brigham on January 26, 1924. This time he had well marked deafness in the left ear and a left facial palsy, indicating an angle tumor.

On February 4, 1924, Dr. Cushing removed nearly completely a left acoustic neuroma, and his general condition has remained very good since that date.

COMMENT: The interesting, although unfortunate feature in this case, so far as our present subject is concerned, was

the attempt on the part of an expert ophthalmologist to differentiate the fundus picture from the type of choking seen in tumor. A measured elevation of 4 diopters could not conceivably be caused by anything but mechanical pressure, a point which was stressed a few years ago by Dr. Harvey Cushing[1].

The following patient was one of the three in the series who were treated on the basis that the condition was luetic. Fortunately, the old idea of a course of mercury and iodides has gradually faded out, so that now, relatively few patients are subjected to delay on this account.

Case III. P.B.B.H. Surg. No. 19941. Gliomatous cyst of left cerebellar hemisphere. Blindness of right eye, and vision in left eye reduced to 5/100 of normal.

Oct. 21, 1923. Admission of Goldie A. A., age 17. The patient's illness began in September, 1922, about one year before her admission, with headaches, accompanied by vomiting, which occurred without relation to meals. Very soon she developed a staggering gait. Because of the increasing severity of these symptoms and the onset of pains in the back and joints, her tonsils were removed in March, 1923. After this procedure, her headaches were worse, requiring morphin for their relief during a period of several weeks. In April, she consulted an oculist because of failing vision and diplopia. He prescribed glasses for hyperopia, but since that time vision failed progressively. During the whole course of her illness, she was given mercury and iodides daily until 2 weeks before she came to Boston.

Examination upon admission disclosed bilateral papilledema of 5 D., with secondary atrophy well advanced. V.O.D. showed light perception only, while V.O.S. was 5/100. Her cerebellar symptoms consisted in a positive Romberg, some moderate ataxia and definite hypotonia.

On Nov. 2, 1923, both Dr. Cushing and the writer participated in an exploration of the cerebellar region. A gliomatous cyst of the left hemisphere containing 40 c.c. of fluid was evacuated, and its lining treated with Zenker's solution. She was discharged on Nov. 24, 1923, and although improved in other respects, her vision remained as it had been just previous to her operation.

Comment: Similarly to Case I, no positive date was given in this patient's record as to the time when her papilledema was first discovered, but presumably this must have been in April,

1923, six months before she was referred to the Brigham, because at that time her vision began to fail and continued to fail progressively thereafter. Furthermore, the advanced atrophy and greatly reduced vision which were disclosed upon her admission, bespoke a process of long duration.

The following patient was also treated for lues, in this instance after choked discs had been discovered, and despite an inconclusive Wassermann reaction.

Case IV. P.B.B.H., Surg. No. 18952. Glioma of left motor area. Vision of both eyes reduced practically to blindness.

June 2, 1923. Admission of Nina E., aged 46. One year before admission, she first complained of headaches, which were soon followed by petit mal seizures. In October, 1922, her vision began to fail, and during the next 5 months she consulted osteopaths and bone setters without relief. In November, 1922, and again in March, 1923, she had convulsions with asymmetry of the face, and in the latter month she consulted an ophthalmologist, who told her that she had choked discs and had a blood Wassermann taken. The latter was reported anticomplementary, but she was given a course of K. I. nevertheless. During this treatment, the ophthalmologist told her that her eyes were improving, but her friends were certain that her vision was diminishing. In May, 1923, she had to stop work because of failure in vision. She then consulted Dr. Blankenhorn, who immediately referred her to Dr. Cushing.

Upon admission to the Brigham, she showed marked mental deterioration, dullness, and disorientation. There was a slight right facial weakness, and tenderness over the left temporofrontal region of the skull. She was having severe headaches. Both pupils were dilated and scarcely reacted to light. She was blind in both eyes, except for bare light perception. The fundi showed bilateral choked discs of 4 to 4½ D. There were large amounts of new tissue over the discs and many hemorrhages radiating from the disc margins.

On June 11, 1923, the writer explored her left motor area and disclosed a tumor of the face center. Four days later, this tumor was removed at a second stage by Dr. Cushing. It proved to be a glioma histologically. She was discharged June 27, 1923. At that time she was relieved of headache and her general neurologic symptoms were improved greatly. Her blindness, however, remained complete, though her discs were flat.

COMMENT: This patient proved to have a type of glioma which was quite encapsulated, and therefore offered a relatively good prognosis after removal. Her usefulness, however, was practically at an end, because she had been allowed to become blind before an intracranial operation was advised.

The following case had an encapsulated tumor which was removed successfully. Fortunately, her vision had suffered no marked impairment, but her focal symptoms were so definite, that it would seem hardly possible that a correct diagnosis should not have been made earlier.

CASE V. P.B.B.H. Surg. No. 19275. Endothelioma of left motor area which had attained the weight of 162 grams before operative removal. Bilateral papilledema of 2-3 D.

July 19, 1923. Admission of Clara J. T., age 48. Fifteen months before entrance she began to have headaches and some mental dullness. In September, 1922 (10 months before admission), she had a convulsion with twitching of the right arm and leg, after which she limped and dragged the right toe, while the right hand felt numb and weak. Four weeks later, she commenced to have vomiting at intervals. She was given the Sippey diet for a month, and her vomiting ceased, but she still dragged her right foot. In February, 1923, she had two more focal convulsions, and in April another period of vomiting. She was told that the latter was a "nervous condition" incident to the menopause. A small growth on the inner side of her lip was noted at this time, and was excised after another convulsion had occurred. She failed progressively with increasing weakness of the right arm and leg, and finally Dr. Hoover was called in consultation. He made a diagnosis of brain tumor and referred her to this clinic.

Examination showed a marked degree of mental dullness and beginning aphasia. There was a right partial spastic hemiplegia, with paralysis nearly complete in the arm. Her fundi showed bilateral choked discs of 3 D., on the right and 2 D., on the left. She could not be made to cooperate in a test for visual acuity. X-ray of the skull showed in the posterior frontal region the typical bony changes overlying an endothelioma.

On July 30, 1923, Dr. Cushing enucleated a large endothelioma from the region indicated. She made an extremely satisfactory recovery and was discharged August 21, 1923, with

strength returning rapidly in the right side. She was able to read ordinary print at this time, but her optic discs, although flat, showed a moderate degree of secondary atrophy.

COMMENT: Here, then, was a patient who had received a common form of symptomatic treatment in the presence not only of severe, but of localizing neurologic manifestations. In her case, no oto-laryngologic problem was involved, and fortunately also her eyes had not been severely damaged. The case is presented merely to illustrate that a long period of delay was allowed, although the evidences of brain tumor were almost unmistakable. Had the neoplasm been one of more rapid growth it is probable that vision would have been distinctly impaired.

DISCUSSIONS AND CONCLUSIONS.

In answer to the questions which were proposed at the beginning of the paper, we may say, that although there are a few conditions other than brain tumor which may cause papilledema, or a condition of the optic nerves simulating it, the percentage of instances in which it occurs in such cases is extremely small as compared to tumor. Second: The previously assumed etiology and treatment of the papilledema in a verified series of 100 brain tumor cases was erroneous in 48 per cent. Third: The delay incident to treatment other than that directed toward the relief of intracranial pressure in these cases, resulted in some damage to the optic nerves in every instance. This damage was severe in 27 patients, of whom several had reached the stage of blindness.

It may be concluded, then, that in the vast proportion of cases, papilledema means brain tumor, or at least a surgical intracranial condition equivalent to it. A neurologist or neurologic surgeon should be given the earliest possible opportunity to see a patient in whom choked discs are discovered, in order that the most frequent as well as the most serious source of this condition may be either ruled out or confirmed by those who are specially trained to diagnose these disorders.

I wish to express my thanks to Dr. Cushing for his permission to report this series of cases from his clinic.

BIBLIOGRAPHY.

1. Cushing, Harvey. Accessory Sinus Disease and Choked Disc. Journ. A. M. A., July 24, 1920, v. LXXV, pp. 236-237.

DISCUSSION OF SYMPOSIUM ON PAPILLEDEMA.

DR. E. C. ELLETT, Memphis, Tennessee: To discuss the mechanism of papilledema means to most of us to recite what we have read of the experiments on the subject, and to endorse the one that appeals to us as the most plausible. Since von Graefe first described what he called "choked disc," in 1860, and attributed it to compression of the cavernous sinus by a tumor, many theories have been advanced and many experiments have been made. The theory most usually accepted is that put forward by Schmidt-Rimpler in 1889, in which he ascribes the phenomenon to distention of the vaginal space of the optic nerve and consequent edema of the lamina cribrosa. The latter element of the theory is not considered important. The fact is, that papilledema is at first a pure edema. The inflammatory phenomena, about which Leber built his irritation theory, are seen later, when the disintegration of the stagnated lymph has furnished a chemical stimulus.

This much you can read in any book. What I especially wish to speak of is the conclusion reached by Dr. Parker in the experiments on unilateral papilledema, which formed the substance of his chairman's address before the Section on Ophthalmology of the A. M. A. in 1916. This work has not received the attention it deserves, and I am glad to have an opportunity to present the history of a case in which his conclusions appear to be borne out.

The patient was a man, aged 44, who was in the hospital with headache and a paresis of the right seventh and right twelfth nerves. The man also had a chronic arthritis. The left optic disc was normal, but the right showed papilledema and swelling of 2 D. Both eyes were anesthetized by three instillations of cocain, and the tension tested with the patient lying in bed. Four readings were made on each eye with a Cohen tonometer. The tonometer was applied by me, and readings were made jointly by Dr. R. C. Bunting, the attending neurologist, and the house surgeon. The readings were as follows: O. D. 16, 16, 16, 17; O. S. 20, 20, 19, 23. The patient died two weeks after this observation was made, and the autopsy disclosed a tumor mass located at about the midportion of the left parietal lobe, approximately 25 x 35 mm. in size, but not sharply circumscribed, merging imperceptibly into the brain substance. The tumor was grayish in color and moderately hard in consistency. At the deeper portion, the tissue affected by the tumor appeared to have broken down, having become soft, degenerated, necrotic, and showing many small hemorrhagic areas. When sections were shaken in water, the "spider" cells were seen.

Stained sections showed the tumor mass to consist largely of spheroidal cells with proportionally large nuclei, a few thin walled blood vessels, and numerous small hemorrhagic areas in the deeper portion of the specimen. Considerable degeneration and necrosis had taken place. Diagnosis: Brain tumor. (Glioma.)

This marked difference, with the low tension in the eye showing the swelling of the nervehead, seems confirmatory, as far as one observation can be, of Dr. Parker's experimental work.

The Cohen tonometer, which does not seem to be generally known, has been, in my hands, by far the most satisfactory tonometer I have

used, in that the readings are more constant. It is a mercury tonometer, and the tension is registered directly by a column of mercury in a graduated tube.

Dr. Hiram Woods, Baltimore, Maryland: These papers are to be discussed from several points of view. I shall try to keep within ophthalmic lights, and present some features of papilledema which have impressed themselves upon me in recent years. A certain amount of overlapping is unavoidable. What any one says must be interpreted in the light of what every one says. The basis upon which this discussion is founded is necessarily the synopsis of each paper as published in the program, and a personal communication from Dr. Bordley.

As stated, papilledema is a symptom. Yet the danger shown by this symptom is often more pressing than the cause itself. The latter may be nonprogressive, or beyond treatment, yet quick blindness may threaten. This touches neurologic surgery, and is the ophthalmologist's getting-off station.

There is ground for believing that some men who ought to know better do not appreciate the meaning of papilledema. The following case is illustrative.

A woman, 42 years old, came under my observation in a hospital for mental disease. She had been sent there for what was called "grave hysteria." I was asked to examine her for glasses. Her symptoms were severe paroxysmal headaches and right sided defective sight. The right nerve was atrophic, pupil slightly dilated without direct light contraction, but preserved consensual reaction. In the left eye, central vision was normal—no reliance could be placed on field examination. Direct pupillary reaction was preserved in the left eye, but consensual was lost, confirming the diagnosis of right nerve atrophy. The left papilla was swollen to 2 diopters, yet before entering the hospital, her physician and at least two ophthalmologists had been satisfied with the diagnosis of "right congenital amblyopia discovered late in life." The head pains were called "hysterical," because general examination did not show a cause. Further study and operative interference disclosed a brain tumor, which was inoperable and eventually fatal. But that does not affect the matter under discussion. The swollen disc with preservation of normal central vision was simply neglected as a symptom of something farther back. Apparently, it was the persistence of good sight in the left eye which led to the conclusion that this eye showed nothing of importance. In view of what Dr. Parker has said about papilledema appearing first in the eye of lesser tension, it is to be regretted that tonometric measurements could not be taken. Lack of cooperation on the patient's part was the reason. To the fingers, tension was normal in both eyes.

The ophthalmologist has an important share in the search for the cause of papilledema. He must correctly, and, as completely as possible, interpret what he sees. The optic papilla may show swelling—choked disc—from what Fuchs calls an "hydrops" of the intervaginal space. This is merely pressure, but it is true choked disc. Again, papilledema may be secondary to the inflammatory exudate in the nerve trunk—optic neuritis. Here, also, the nerve swelling is the result of pressure, but the source of that pressure is in a different place and usually from a different

underlying cause. Ophthalmoscopic appearances of these two conditions are characteristic enough in typical cases. An elevated disc, without blurring of the neuroretinal margin or broadening of the veins or small hemorrhages, forms an easily recognized picture; but it is not a long step from this condition to minute fundal hemorrhages and haziness of the neighboring retina, as shown by Marcus Gunn's classification. This at once brings us up to a possible neuritis.

Dr. White has given us a valuable point in diagnosis, when he speaks of the vision disturbances in neuritic edema. This diagnosis is of immense importance because of the difference in causation. The hydrops edema points to increased intracranial mechanical pressure; the inflammatory edema suggests infection. The somewhat acrimonious discussion of recent years about the relation of sinusitis and other infections to choked disc doubtless has its origin, at least in part, in failure to appreciate optic neuritis as a cause of papilledema. A great deal of this confusion is removed by the lessons of these papers. Dr. Bordley says, "choked disc is always dependent upon an increase in intracranial pressure." This is exactly what Harvey Cushing has maintained. There is nothing new about it; it is simply rescuing the truth out of a fog largely of our own making. Dr. White is perfectly right in advocating a new term for disc edema associated with sinusitis and other infections. The suggestion of "optic neuritis with edema" will do till we get a better term. "Neuritis edema" is a little shorter and seems to me as clear. Dr. White gives a sound bit of advice, when he protests against exenteration of accessory sinuses before a thorough neurologic examination. Put in another way, this means: Come to no fixed conclusion as to cause till every possible etiologic factor has been studied, no matter how positive pointings may be in any one direction.

Finally, the ophthalmologist must keep track of ocular conditions which might aid in diagnosis. Now and then there is a false alarm. Such a simple thing as ametropia sometimes produces neuroretinal appearances suggestive of papilledema. The oculomotor structures sometimes develop changes valuable in localization. The same is true of perimetry, field contraction, scotomata, enlargement of the normal blind spot, and so forth. Dr. Bordley thinks field study offers "but minor advantages." Maybe so, when compared with ophthalmoscopy or neurologic history and findings; but it will not do to endorse this to the extent of excluding repeated perimetric tests; there are too many possibilities to justify this. One of them is localization.

In a word, papilledema is but the beginning of a diagnostic study which may take us anywhere, and we must follow step by step. The ophthalmologist must keep up with his own responsibilities, but he cannot solve the problem alone.

Dr. G. E. de Schweinitz, Philadelphia, Pa.: Whether one believes that choked disc, or papilledema, is due to lymph stasis, or to this condition plus venous engorgement, as Paton and Gordon Holmes maintain, or to passive lymph stasis depending upon interruption of the lymph passages of the optic nerve passing centrally (Carl Behr), the fact remains that the activating or predominant factor, as Bordley has said, is increased intracranial tension; if other factors are potent, they have not been discovered.

We cannot avoid dealing with types of nervehead changes which have other origins and justify the various terms, "optic neuritis," "inflammatory optic neuritis," "toxic optic neuritis," "optic neuritis with edema," and so forth. In the early periods of the two conditions, a distinction or differential diagnosis is often difficult, and obviously it cannot be made merely according to the degree of swelling which is present, 2 D. or more characterizing choked disc, as has been maintained by certain ophthalmic surgeons. Hence, it is desirable to extend the observations of Koeppe, who, using the Gullstrand diaphragm lamp, with special apparatus and illumination, finds that the anterior vitreous is normal in the early stages of choked disc, while in optic neuritis, whitish, defined lymphocyte flakes are visible; and that in incipient choked disc, the papillary structure is visible, but in inflammatory optic neuritis, it is clouded or obliterated.

It is also occasionally admittedly difficult to distinguish the socalled "pseudoneuritis" or "spurious optic neuritis," as it occurs in hyperopic and highly astigmatic eyes, from the early stages of papilledema and papillitis, in spite of ophthalmoscopic distinctions. In such circumstances, careful perimetry furnishes the more exact method, particularly as applied to the detection of alterations in the size of the blind spot, or more exactly, the scotomatous defect which surrounds or joins it (Traquair) in beginning papilledema, and the reaction to graded test objects, colored and white. When there is an excess of choking of the disc in one eye as compared with its fellow, as is commonly the case, ophthalmoscopic measurement is usually sufficient to determine the difference. But not always, especially in the case of slight degrees; also, it may be desirable to ascertain which eye presents the older process, that is, in which eye the process first appeared. Parker's test is available, but I am practically sure a careful analysis of the socalled enlargement of the blind spots by approved modern methods will yield information by which this point can be determined.

Also, as we know, in tumor or abscess of the frontal lobe, there may be papilledema in one eye and no disc swelling in the other eye, but an axial retrobulbar neuritis which is ipsolateral to the lesion. The advantage of visual field testing is obvious. Therefore, although it is quite true that perimetric examinations do not develop symptoms which are characteristic of choked disc, they are far from useless in the study of this condition.

Although the papillitis and socalled papilledema which depends upon infections in the accessory sinuses, and sometimes on focal infections in the teeth and tonsils, differs from that produced by increased intracranial tension, as Dr. White points out, it must not be forgotten that examination of the fundus in paranasal sinus disease, particularly ethmoiditis and sphenoiditis, may sometimes disclose very high degrees of disc swelling which ophthalmoscopically very closely resemble choked disc, which are due to a combination of factors, venous stasis, pressure and toxemia. Perhaps, as previously mentioned, slit lamp investigations would be worth while in making a differential diagnosis, but just because occasionally these intense degrees of swelling occur, and also in any case of more moderate disc change, it is eminently proper, as Dr. White so clearly insists, that a careful neurologic examination should precede any interference with the sinuses from the surgical standpoint.

It is, perhaps, not inappropriate to say one word in regard to the scotomas which find their habitat in the central part of the field in various types of sinusitis. They may be pericentral, or, as it is usually called, central, or paracentral, and sometimes they appear in the centrocecal form. Now, in a certain number of the cases of sinusitis optic neuritis, or to use White's term, optic neuritis with edema, central, or better, pericentral and other types of scotomas are associated phenomena, and yet these scotomas of themselves do not present features which are characteristic of sinus disease, and they must be studied in association with all other portions of what may be called the clinical picture. As Dr. Traquair has recently said, one finds frequent allusions in the literature to the field changes which resemble those due to sinus disease, and statements that they may occur with pituitary body tumor, tobacco poisoning, and even glaucoma. In these circumstances, we surely can by differential and analytic methods of perimetry, not to mention other methods of examination, differentiate the scotomas, for example, the "quadrant type of scotoma" in chiasmal interference, the distinctive features of the tobacco scotoma, and those of the glaucoma scotoma, and therefore a confusion such as these references in literature bring about could not occur.

Dr. Woods and Dr. Horrax referred to the mistakes that are made and the unnecessary interference surgically, when really there is intracranial tension which is the activating factor. This is perfectly true, and I want only to add my emphasis to that brought out this morning. I have, myself, seen a double choked disc in a patient who was just about to have all her teeth pulled, but happily she died quite suddenly, and autopsy showed that she had a tumor as large as a hen's egg in the silent area of the brain. That was a case in which snap diagnosis led to what would have been a sad mistake. But I have also seen a high degree of optic neuritis, especially with astigmatism and hyperopia, with a good many symptoms that suggested brain tumor, with the patient practically prepared by the surgeon, when the correction of about five diopters of astigmatism made a well woman of the patient, and she is a well woman today. So let us by all means, as Dr. Woods says, take into consideration all the factors in these cases. But it is not quite just to say that all the indiscretions of diagnosis are ophthalmologic or otologic; they are also occasionally surgical and neurologic.

Dr. P. E. Bousquet, Montreal: After the elaborate lectures we have heard this morning, there does not seem to be much more to say on the subject. However, allow me to mention a few points in regard to the influence of (a) lues, (b) traumatisms of the skull, even noncomplicated, and (c) oxycephaly.

In mentioning luetic infection, I mean to speak of lués precox. Cantonnet, in 1912, observed a series of cases of papillary stasis accompanying luetic headaches in the formerly called secondary period. Even at that time, Cantonnet had noticed that recent luetic patients suffering from intense headaches had stasis. We may read the history of these cases in the thesis of his assistant, Fombeure (Thesis of Paris, 1912). In 1914, Lavaut found in luetic patients presenting cephalgia, an increase of the tension in the liquid.

The idea of stasis in traumatism of the skull is of recent date. Professor de Lapersonne and Dr. Velter have met it in civil practice

(Annales d'Ophtalmologie, April, 1913). Cantonnet relates the observation of 18 cases of traumatism of the head, 10 of whom had slight papilledema, some of which were monolateral. Lumbar puncture showed hypertension and produced amelioration. Sight improved, headaches and dizziness disappeared. In 2 cases, veins of retina were tortuous and dilated, with normal disc edges and no true stasis. All these patients were coming from a neurologic center, of which Henry Claude, well known French neurologist, was chief. Dautrelle (Archives d'Ophtalmologie, May and June, 1916) also reports some interesting cases. In the luetic infection of the envelopes, as well as in the cranial traumatisms, Cantonnet contends that we may find all the degrees of stasis, from the simple stasis of edema. He shows some cuts, in which we can see the distention of the subarachnoid spaces.

I wanted to point out the influence of oxycephaly on the development of papilledema, but my good friend Horrax mentioned it this morning, and I will only mention the evolution of these papilledema either from precox luetic meningitis infection, or secondary to cranial traumatism, or from oxycephaly.

In neither case, unless we search for it at the very beginning, do we see the patients early enough to assist at the complete evolution of the disease. This, I believe, is the reason we at times meet cases of atrophy of the optic nerve without any other history than that of a trauma of the head, as from a fall, or of any other kind. I had the opportunity of observing a case of oxycephaly in full atrophy, and the pathologic changes around the disc and vessels, together with the statistics of 60 cases studied by Patry (Encyclopedie Française d'Ophtalmologie, Vol. VII) induced me to come to the above conclusion.

In conclusion, we should always bear in mind, that at the posterior pole of the eye, there is a manometer where we can read and appreciate the level of intracranial pressure on patients suffering from headaches. This manometer observation, if made a matter of routine, will enable us to begin proper therapy at the proper time, and so do much to prevent many cases of fatal blindness.

Mr. Chairman, I wish to express to you and to your committee my most cordial thanks for the honor you paid me in inviting me to take part in the discussion of this very interesting question of papilledema, and to thank you also for the opportunity of hearing and appreciating the masters of American ophthalmology and oto-laryngology.

Dr. Adelbert Fuchs, Vienna: I wish to make a few remarks about the most interesting paper of Doctor Parker, and his excellent experiments on the etiology of papilledema. I wish also to emphasize that I believe in the mechanical etiology of papilledema, but the explanation is very difficult, because we find so many details which are opposed to this mechanical theory. The theory of Schmidt-Rimpler, of hydrops of the sheath of the nerve, is not always possible. Klauber showed that in a certain number of cases where choked disc was to be seen during life, postmortem showed no fluid in the vagina and no hydrops of this vagina. And so general edema of the brain may be contained in the optic nerve and in the head of the optic nerve. On the other hand, it is known that in some cases of brain tumor, postmortem examination, or examination during the operation, does not show an edema of the brain; but there

are cases where the edema is very marked. In other cases, where choked disc is present, there is no edema.

Another great difficulty in mechanical explanation is the fact, that the edema disappears when the optic nerve becomes atrophied. We all know that in old cases of tumor of the brain, when the patient is blind and the optic nerve is quite white and atrophied, there is no swelling of the head, in spite of the fact that all the vessels are present, so that edema should be expected. I cannot explain this occurrence, but Karl Baer, in one of his papers on choked disc, brought out a very interesting explanation. It would be too long to speak about here.

I wish to point out only two other things. We know that there are some brain tumors where a very well developed choked disc is found in one eye and no choked disc in the other eye. It may be that these cases are to be explained according to Doctor Parker's findings—that one eye is softer than the other. These cases were not examined with the tonometer. So we have cases of brain tumor with monocular choked disc.

Then, we have the possibility of papilledema with the clinical aspect of choked disc, where the disc is closed by tumor, not in the brain, but more in front. If I may show a schematic picture (illustrates on blackboard) of the posterior pole. There are cases, where an infiltration by sarcoma cells is found surrounding the papilla in the choroid. The choroid would be here, and here would be the retina. I am sorry that I could not bring such a slide with me. I have such a slide in my collection, but I have sent it to New York. Cyst sarcoma is in the beginning a very thin layer and the clinical picture is that of choked disc. It is very difficult to make a diagnosis, but later on the anterior glioma gives an explanation.

Another possibility for choked disc we see behind the eyeball. I saw such a case where there was an exophthalmos. This was not very considerable, but there was a tumor behind pressing not so much against the bulb, as against the optic nerve, and the optic nerve was compressed by the growing tumor, and choked disc was found clinically.

The greatest difficulty in our work is, that we cannot differentiate so easily between the inflammatory and the congestive form of the optic changes. Therefore, in each case, we should make special examination of the body and consider all the points possible.

Dr. C. Norman Howard, Warsaw, Indiana: I would like to emphasize three gross points of differentiation that have been made, especially by Dr. White, between the socalled papilledema due to accessory sinus, and that due to brain tumor.

It would seem from what we have heard, that for the most part, in the brain tumor, the effect on the eye is, first, slow; second, practically always eventually bilateral; and third, nothing will be of permanent help that does not include the ultimate return of normal brain pressure. On the other hand, involvement of the nervehead growing out of an accessory sinus disease is, first, more rapid; second, it is more apt to be unilateral; and third, its relief lies alone in action against the offending sinus.

In 1923, I reported a case of papilledema to the Indiana Academy (Accessory Sinuses of the Nose in Relation to the Eye. Transactions of the Indiana Academy of Ophthalmology and Oto-Laryngology, January

17, 1923), which happens to illustrate these three points. First, the loss of vision in the right eye from normal to total blindness was three weeks; second, the sight and the disc in the left eye were normal then, nor have they ever become otherwise; third, after I operated on the right sphenoid sinus, there followed a slow, progressive return of sight, until after about nine monhts, it had returned to practically normal. No brain symptoms developed at the time, or since.

Dr. Allen Greenwood, Boston: We have heard a good deal this morning about the degree of elevation of the disc, ranging from 2 to 8 diopters. I would like to call attention of the members to the fact, that in measuring the elevation of the disc, we must be very expert in measuring refraction with an ophthalmoscope. To measure the depth of the eye in the various meridians and locations is absolutely necessary, if one is going to get an accurate measure of the elevation of a disc.

Some time ago, I was called in consultation to see a man who was to have an operation for a brain injury because he had a papilledema. The measurement, as given by those who had measured the disc, was 5 diopters. I found in this case a man with 4 diopters of hypermetropia, which was not considered when the disc was measured. As a matter of fact, the disc was elevated 1 diopter or less. An operation was advised against, and the patient promptly recovered. When I hear of papilledema or optic neuritis combined with papilledema, which comes from sinus disease, with an elevation of 8 diopters, I am inclined to question whether that is an accurate measurement.

Dr. Robert Scott Lamb, Washington, D. C.: I would like to suggest the point that we do have brain tumors with no papilledema at all. I have three cases in mind (every one in Cushing's hands); one was an hypophyseal tumor, one was a cerebellar tumor, and the other was an acoustic tumor. There was absolutely no swelling of the nerveheads in any of these cases.

Dr. V. A. Chapman, Milwaukee: There is one point that has not been brought out, which I wish to mention, with reference to the amount of swelling of the nervehead. The same amount of springing forwards of the nervehead, as measured by diopters, does not always indicate, in different individuals, the presence of the same amount of edematous process. We must take into consideration the size and depth of the physiologic cup as it existed in the individual case before the edematous process occurred. I have seen cases where the edematous process has gone on to considerable extent before any swelling forwards of the nervehead is manifested. These are cases in which there was normally a large, deep physiologic cup before the pathologic process began.

The physiologic cup must be closed by the edema and disappear, before springing forward of the nervehead can occur. Of course, one thinks of looking at the other eye, to compare the appearance of the physiologic cup on that side, and this often aids in our estimation of the amount of edema present. But we must remember that the edematous process may be occurring in that eye at the same time, to a possibly lesser extent.

Dr. Leon E. White, Boston, (closing): Dr. Skillern wished me to express his views on this subject, and wrote out the following: "I

believe that papilledema may occur from an accessory sinus infection, but that it is transitory and represents an early stage of an optic neuritis."

I agree perfectly with Drs. Bordley and Horrax. I have quoted a good deal in this paper, because I thought the expressions of the different specialists who had written on the subject should be brought to your attention. Persistent papilledema is, I believe, practically always due to increased intracranial pressure. I refer such cases to the neurologist or the neurologic surgeon. My cases were only three out of 75, were all transitory, of short duration, and the elevation of the disc was not over two or three diopters. The probability in all cases of papilledema is some intracranial disturbance, and I think it is up to the rhinologist to take the burden of proof upon himself, and prove there is not some neurologic condition back of the papilledema before he advises opening the accessory sinuses. The question has been asked, if it is ever advisable in brain tumor to operate on infected sinuses, teeth or tonsils. Should these infections be producing marked impairment of the health or interfere seriously with the patient's comfort, one might be warranted in doing enough to, at least, afford temporary relief.

As to Dr. Greenwood's remarks concerning the elevation of the disc, I simply quoted the report from Sluder and others. I have not seen those cases. If a case were sent to me with an elevation of 4 to 8 dopters, I should think it was due to intracranial disturbance, and refer it to the Brigham Hospital.

Dr. Gilbert Horrax, Boston (closing): I think we are all in virtual agreement on this subject. I want to express my appreciation of all the things that I have learned at this meeting in the various discussions. I have been very much interested in the opinions as to the differentiation between optic neuritis and papilledema, or choked disc. We have had the usual trouble in differentiating between these conditions in Dr. Cushing's clinic, and we have come to the conclusion that it is a thing you can at best only guess at, by the means which already have been assigned, namely, that in optic neuritis the loss of vision is apt to be more rapid and the increase of the blind spot greater than in true choked disc. However, we have had a good many cases, especially those which we termed arachnoiditis, which are undoubtedly inflammatory, but nevertheless cause increased pressure, in which the disc gives all the appearances of choking, but with very rapid diminution in vision. In that type of case, we feel there is the greatest difficulty in differentiating between the two conditions.

So far as the position of the neurologic surgeon is concerned, I do not want to assume that we do not make mistakes, because that is far from the truth; but I simply wanted to bring out the discussion on this point, and refer to a specific series. The 100 cases studied were all verified tumors, and all had choked discs. We know that a certain number of tumors do not have choked discs, but with such we have not dealt in this report.

TRANSACTIONS

OF THE

TWENTY-NINTH ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

OPHTHALMOLOGIC DIVISION

NONOPERATIVE TREATMENT OF CATARACT.

ALLEN GREENWOOD, M.D.

BOSTON.

In searching the literature pretaining to this subject, one is early impressed by the diversity of the methods employed, the lack of conclusive data, and the differences of opinion as to the merits of the various procedures. When one considers the nature of cataracts, their propensity for various rates of progress, and their frequent periods of remaining stationary, it is not to be wondered at that it has been difficult to judge fairly as to the efficacy of any treatment; but, the intuitive sense that comes to those who have for many years treated their patients with incipient cataract that a good deal is being accomplished, while not of value statistically, cannot be ignored. In order to present the subject more clearly, let us consider for a moment what the term cataract as applied to the eye means to the average ophthalmic surgeon and also to the average layman.

To the ophthalmic surgeon, any opacity occurring in the lens or its capsule may receive the title of cataract. It may be a congenital opacity or be acquired at any time after birth. The opacity may be in any part of the lens, and, after its discovery, may be stationary or progressively increase. Those which progress steadily or intermittently are those occurring most frequently after middle life, and are generally classed under the term senile. In passing, it may be mentioned that the lens viewed through the dilated pupil with the corneal microscope aided by the slit lamp will almost universally show some specks or opacification, but only such opacities as are visible to the observer who uses oblique illumination and the ophthalmoscope for their detection should be entitled to the term cataract.

To the average layman, however, the term cataract usually implies some almost malign growth, which will soon bring on blindness and necessitate a serious operation for its removal, with some uncertainty as to the end results. He perhaps recalls some elderly parent or grandparent who was

made blind by cataracts, and whose vision was not restored by an operation which perhaps required much time motionless in bed in a darkened room. The term cataract certainly strikes terror into the soul of some of the timid ones, and, in my opinion, *this term should be used only very sparingly to one's patients.*

The practice of using the word cataract only when the opacities are nearing the stage of completeness, or when an operation is soon to be expected, is a good one to follow. The older custom of announcing to the patient who has a few lenticular opacities that cataracts were present, and that nothing could be done until they were "ripe" is, fortunately, going out, while the custom of giving patients with incipient cataracts good counsel and careful treatment is coming in. What, then, constitutes good advice and careful treatment, and what may we expect to accomplish? This brings us to the discussion of the nonoperative treatment for incipient cataract. This treatment may be roughly divided into four varieties. First, the hygienic; second, the use of local applications; third, the use of electricity, radium, and the X-ray; and fourth, the use of lens antigen.

Under hygienic treatment is included all the efforts to improve the body metabolic processes, improvement in the cardiovascular system and the blood. In this latter, particular attention should be directed to the reduction of any abnormally high blood sugar content; and here let me suggest to those critics who claim never to have seen any good results from the nonoperative treatment of cataracts, that their experiences cannot have included many cases of incipient cataracts due to diabetes, or included cases apparently due to foci of infection. Under hygienic treatment, also, comes the advice we give our patients in regard to the use of the eyes and as to the avoidance of any undue eyestrain.

In the prevention of eyestrain, too much stress cannot be laid on the necessity of properly fitted and adjusted glasses, with fairly frequent retestings. Reassuring the patients and encouraging them to hopefully follow out the treatment, has a mental effect not to be ignored. That the hygienic treatment has alone been of great value in the hands of some of our best observers, is amply attested by the writings of Risley in 1891, and more recently by Jackson, in his paper read before the American Ophthalmological Society last June.

In regard to the effect of local applications, more has been written than about any of the other forms of treatment. A variety of drugs in solution, dropped into the eye or injected subconjunctivally, have their ardent advocates. From a description of the use of these various solutions, one finds that the advocate is depending on their action to increase the circulation and, hence, the nutrition of the anterior portion of the eye, or is depending on their antiseptic action, or on both. Of all the drugs, "and their name is legion," one, on account of its peculiar action on the lymph circulation of the anterior portion of the eye has come to be used more than any other; namely, dionin. In using dionin, one must never disregard the fact that the lymph vessels soon become habituated and do not respond to the treatment. Therefore, to get the desired effect repeatedly, the drug must be used intermittently. This is often forgotten, and dionin ordered to be used continuously, with the result that only disappointment will follow from the treatment. To get a greater effect, dionin is sometimes used subconjunctivally. Of other drugs suggested, those most frequently mentioned are iodid of sodium or potassium, fibrolysin, nascent calcium iodid, iodolysin and tinctur cineraria maritima, and for subconjunctival injection biclorid or cyanid of mercury or dionin. Some observers who have used the cyanid of mercury injections have reported very good results from this method of treatment.

Of the treatment by electricity, radium and the X-ray, that by electricity has been the most exploited of any of the methods of nonoperative treatment of cataract. The former custom on the part of ophthalmic surgeons of offering no treatment to their cataract patient, resulted in such patients turning to men who offered to cure all cases of cataract by the use of electricity, and "thus avoid the danger of an operation ever being necessary." Some of these "cure all men" used a weak solution of atropin with each treatment, to get some improvement in vision due to the enlarged pupil, and thus encourage their victims to come back for more electricity. In the hands of competent observers, electricity, applied in various forms and ways, has not proven of much value. For a while, the use of radium seemed to promise much, and its application was carefully worked out by Cohen and Levin, and Franklin and Cordes, who reported encouraging results, but the test of time has already lessened the optimism of its advocates.

Recently, the use of lens antigen has been advocated, and some most encouraging results have been reported by the leading exponent of this treatment, Dr. A. E. Davis. In studying the cases reported by him, one finds that those included among the early ones apparently have secured benefit, and the growth of the opacities has been retarded, at least for some time. The later cases, which form the larger number, are too recent to base acceptable deductions on, and, while some have shown improvement in vision and a lessening of some of the opacities, to claim that no future increase will take place in the opacities will only be warranted after a goodly number of years of careful observation of all the cases. The large number of injections found necessary in order to get results must be disturbing to some of the elderly patients. He, however, who would unthinkingly claim that this treatment with lens antigen will never prove decidedly helpful, may some day find he is in the minority. Further experimentation along this line by others may give gratifying results.

Having briefly noted some of the methods employed in the nonoperative treatment of cataract, let us consider for a moment what the ophthalmic surgeon has hoped to accomplish and what seems, on a review of the literature, to have been accomplished. We have all, at times, desired in some way to improve the clearness of vision in our cataract cases and, above all, in the very incipient cases, to stop the increase in the opacification. The great obstacle, however, to a clear working out of the problem is that the desired results frequently come about when no treatment at all has been applied. In spite of this, there are very many observers who claim that at least a partial accomplishment of the desired result is much more common following treatment than with the no treatment plan.

While some still express the belief that treatment is of no value, there are probably very few today who send patients with vision blurred by lenticular opacities away without any treatment. The accurate adjustment of correcting lenses is in itself a valuable method of retarding the development of the opacities.

There are few today who, suspecting the presence of diabetes, do not advise treatment of the underlying cause, and who have not found an improvement in vision and an

arresting of the process following a cessation of the glycosuria brought about by diet or insulin or both.

There are few today who, suspecting and finding some uveal disturbance, do not treat the underlying causes and note an improvement resulting.

We are all, therefore, wittingly or unwittingly, applying a nonoperative treatment for our patients who have come to us for relief of a blurred vision due to lenticular opacities.

Many ophthalmic surgeons, besides using the practically universal hygienic method, are in addition using some of the other special methods, with the conviction that something is being accomplished. Many of the observers, who, like the author, feel that special treatment as well is of value, have found that certain types of opacity are more helped than others. A simple clouding of the nucleus is often not helped, and this is also true of the radiating or wheel spoke lines of peripheral opacification. The cloudiness between the radiating lines is most often influenced and frequently disappears under treatment.

During the first 10 to 15 years of the author's practice, no nonoperative treatment was given cataract patients; but during the past 20 years consistent efforts have been made along this line. During the whole of the later period the author has carefully carried out the hygienic measures, and during the bulk of the time has consistently carried out the intermittent use of dionin. Sometimes the dionin is given to be used alternate weeks; but more often, to be used two nights each week, Saturday and Sunday being usually chosen as most convenient for the patient. The drug is used in a 1 per cent solution, which may later be strengthened if necessary.

What have been the results? In the types most often mentioned as being influenced, there has been, in a good many cases, a definite clearing in the lens as viewed with the ophthalmoscope, and a corresponding improvement in vision. A careful drawing of the opacities is made at the first examination, and new ones made as the opacities change. Frequently, a vision of 20/70 is bettered, so that 20/40 or even 20/30 is easily demonstrated. Such an improvement has only rarely been seen under the no treatment plan. Such an improvement has frequently been maintained through a number of years, even to 5 or 10 years. An improvement

in vision and a retarding of the progression is more likely if, coincident with the use of the dionin treatment, there is an improvement in the general condition, with an elimination of all foci of infection or abnormal blood states. It is therefore plain that general treatment should be combined with any form of local.

In a recent excellent paper, read by Dr. G. F. Harkness before The Iowa State Medical Society, 1924, on "Incipient Cataract," there is much food for thought. He incorporates in the paper the answers received from a questionnaire sent to those occupying the chairs of ophthalmology in a number of the class A medical schools of the country and tabulated the answers. Those who claimed that no line of treatment had been successful, and those who claimed that some treatment was positively or at least possibly helpful, were about evenly divided. It is interesting to note, on looking over the answers, that of those who had found that some local treatment was apparently of help, the majority favored dionin. The majority of those emphatic in the statement that no treatment was of use, did not state that they personally had given any of the most generally used methods a long and carefully studied trial. Such a study of a method requires many years and not a few months. For a detailed description of the multitude of methods used in the nonoperative treatment of incipient cataract one should study the literature or read a resume such as is included in the paper of Dr. Harkness, for it is not the purpose of this paper to present all the methods and supposed results in detail. The object of this communication is accomplished if it influences some to have more hope, that gradually out of the darkness there may come more light for us and our patients.

DISCUSSION.

DR. F. W. DEAN, Council Bluffs, Iowa: My treatment of incipent cataracts has been limited to a single subconjunctival injection of twenty minims of a 1 to 5000 solution of cyanid of mercury, and I have used no subsequent treatment, either local or general. I make the examination with the ophthalmoscope and focal illumination, and make a note of the appearance of the lens, then record the best vision that can be secured with correcting lenses without the use of a mydriatic. At each examination subsequent to the injection, the same procedure is followed.

The technic of the treatment is as follows: Patient is anesthetized with morphin, atropin and scopolomin, then, with a fixation forcep, the conjunctive is grasped one centimeter from the margin of the cornea, and between the superior and external recti muscles. A hypodermic needle is passed through the conjunctiva near the grasp of the forcep, and the solution injected well toward the posterior portion of the eye.

Colonel Smith says that the injection must be given when the first haziness of vision is noticed. While it is true that the treatment should be given early, I have found that much improvement may be expected even after the vision has become decidedly blurred. The cataracts I have treated are of two types, or of three, the third being a combination of the other two: The nuclear type, manifested by a sclerosis and haziness of the lens nucleus. The cortical type, showing either subcapsular striae extending from the equator toward the center of the pupil area, or fine dot like opacities and a general haziness of the lens cortex. In cases 1 to 100 treated in this way, I was able to secure reexamination of 129 of the 164 eyes injected.

I have arrived at the following conclusions: A large majority of cortical cataracts of not too long standing are distinctly improved by the subconjunctival injection of cyanid of mercury. In cataracts purely nuclear in type, the vision is not improved by the injection, though I am not prepared to say definitely that the progress is not retarded. Cataracts of the combined nuclear and cortical types are improved only to the extent that the cortex clears. The injection of one eye in an advanced case is helpful to the patient, while waiting for the cataract of the second eye to mature to the operable stage. I cannot believe that those who say they know of nothing that will benefit an incipient cataract or improve the vision, base their reports upon a thorough trial of all the proposed remedies or a thorough study of the results obtained from the treatment.

I wish now to show a few slides which are records of some of the cases I have treated:

V. improved in 88 of 129 eyes, remained unchanged in 34 reexamined, continued to fail in 7.

Cortical. V. improved in 60, remained unchanged in 11, continued to fail in 2.

Nuclear. V. improved in 21, remained unchanged in 22, continued to fail in 4.

Combined. V. improved in 7, remained unchanged in 1, continued to fail in 1.

In the following slides, the line under the record of vision indicates that the injection of cyanid of mercury was made on that date.

Case 3. Mr. J. A. B. (63). July 26, 1921. Bilateral cortical cataracts; cortex contains fine dot like opacities with a general haziness—the type we expect to develop rapidly.

Vision (corrected)

	R. E.	L. E.
July 26, '21	20/30	20/50
Aug. 5, '21	20/30	20/30–4
Oct. 8, '21	20/40–2	20/20–1
Feb. 11, '22	20/20–3	20/20
Dec. 16, '22	20/20–3	20/20
Jan. 17, '24	20/20–3	20/20–2
Aug. 10, '24	20/20–1	20/20

You will note that the vision of the left eye improved after the injection on July 26th, while the vision of right eye continued to fail until it was treated on October 5th, after which the vision improved; and three years after the first injection, the vision of each eye is normal.

Case 97. Mrs. H. A. D. (74). March 20, 1924. Incipient cortical cataracts.

Vision (corrected)

	R. E.	L. E.
Mar. 20, '24	20/50	20/50
Apr. 3, '24	20/40–4	20/40–3
Apr. 12, '24	20/30–1	20/30–3
Apr. 24, '24	20/20–4	20/20–3
May 10, '24	20/20–3	20/20–1
July 12, '24	20/20–4	20/20–2

This patient is a sister of case 3, with the same type of cataract. I show this slide to show the rapidity of improvement following the injection in this type of cataract.

Case 5. Miss M. E. S. (66). Aug. 2, 1921. Bilateral nuclear cataracts with subcapsular striae in each lens. Cataracts are of some two years' duration.

Vision (corrected)

	R. E.	L. E.
Aug. 2, '21	20/50–3	20/50–3
Sept. 19, '21	20/50–1	20/50–2
Oct. 10, '21	20/40–2	20/40–3
Nov. 15, '21	20/40–3	20/40–3
Oct. 7, '22	20/40–3	20/40–2

A decided improvement, but due to the clearing of cortex. This case, No. 5, was an early case. A patient with bilateral nuclear cataracts, with vision in each eye less than 20/50, would now have only one eye treated.

Case 6. Mrs. F. W. W. (50). August 2, 1921. Vision failing a few months only. Cortical cataracts in each eye with subcapsular striae extending entirely across the pupil area.

Vision (corrected)

	R. E.	L. E.
Aug. 2, '21	20/20–6	20/50–2
Aug. 10, '21	20/30	20/50
Sept. 1, '21	20/20–2	20/40–4
Jan. 25, '22	20/20–2	20/30–1
Dec. 16, '22	20/20–3	20/30
Jan. 9, '24	20/20–2	20/20–4
Aug. 8, '24	20/20–3	20/20–3

The cortical striae still extend across the pupil area, but the spaces between them are clear and allow good vision.

Case 7. Mrs. C. W. M. (66). August 3, 1921. Bilateral nuclear cataracts.

Vision (corrected)

	R. E.	L. E.
Aug. 3, '21	20/30	20/50
Aug. 13, '21	20/30	20/30
Sept. 19, '21	20/20–3	20/20–3
Dec. 14, '21	20/20–2	20/20–3
Apr. 21, '22	20/20–4	20/20–2
Apr. 23, '23	20/20–2	20/30
Mar. 11, '24	20/20–3	20/30

A haziness of left lens was undoubtedly cortical though no markings were visible.

Case 60. Mr. W. W. F. (46). Oct. 5, 1922. Photographer. Incipient bilateral nuclear cataracts plus cortical striae.

Vision (corrected)

	R. E.	L. E.
Oct. 5, '22	20/30–1	20/30–1
Nov. 2, '22	20/30	20/30
Jan. 2, '23	20/30	20/30
Dec. 28, '23	20/70	20/50
July 15, '24	20/200	20/70

The correcting lens has changed from —0.50 on Oct. 5th, '22, to a —5.50 on July 15th, '24.

Case 80. Mrs. E. E. F. (60). Feb. 15, 1923. Bilateral cortical cataracts. Four striae in right lens and twelve in left.

Vision (corrected)

	R. E.	L. E.
Feb. 15, '23	20/20–2	20/20–4
Mar. 21, '23	20/20–3	20/20–3
Aug. 13, '23	20/20–2	20/20–4
Feb. 14, '24	20/20–1	20/20–3
Aug. 7, '24	20/20	20/20

No striae in right lens; three on nasal side, faintly seen, in left. Disappearance of striae in this case, among others, convinces me that striae are visible before there is actual degeneration of the lens fibers.

CASE 47. Mr. A. J. W. (71). March 8, 1922. Bilateral cortical cataracts. Fine subcapsular striae and a general haziness.

Vision (corrected)

	R. E.	L. E.
Mar. 8, '22	20/40	20/40
Mar. 28, '22	20/30	20/30
Apr. 19, '22	20/30–2	20/30–3
May 17, '22	20/40	20/20–3
Dec. 16, '22	20/20–4	20/20–3
May 22, '24	20/30	20/20–4

CASE 34. Mr. B. A. G. (64). Dec. 17, 1921. A locomotive engineer. Bilateral cortical cataracts. Must get better vision or give up his engine.

Vision (corrected)

	R. E.	L. E.
Dec. 17, '21	20/20	20/70
Jan. 6, '22	20/20	20/40–1
Feb. 7, '22	20/20	20/30
May 18, '22	20/20	20/30–2
Nov. 13, '22	20/20	20/30–2
Jan. 21, '24	20/20	20/40

He still has his engine.

CASE 22. Judge A. S. W. (74). Oct. 10, 1921. Bilateral nuclear cataracts; left vision mature. Injected right eye to hold the vision.

Vision (corrected)

	R. E.	L. E.
Oct. 10, '21	20/40–4	Objects
Dec. 28, '22	20/40–3	
Nov. 26, '23	20/40–2	
Nov. 27, '23		Operated L. E.

Vision of right, treated eye remained unchanged for two years, while left cataract matured.

CASE 19. Mrs. D. W. K. (70). Oct. 4, 1921. Bilateral cortical cataracts; eyes have only recently begun to blur.

Vision (corrected)

	R. E.	L. E.
Oct. 4, '21	20/20–5	20/30
Nov. 9, '21	20/20	20/20–4
Oct. 17, '22	20/20	20/20–2
Jan. 8, '24	20/20–3	20/20–5
Aug. 7, '24	20/20–4	20/20–6

Nearly three years have elapsed, with no progress, but rather a slight clearing of vision.

CASE 44. Mrs. A. E. B. (59). Mar. 3, 1922. Bilateral cortical

cataracts. There is a haziness of the cortex; right lens has one subcapsular stria. Has sensation of veil before eyes.

Vision (corrected)

	R. E.	L. E.
Mar. 2, '22	20/20–1	20/20–2
Apr. 12, '22	20/20–1	20/20
(Impression of veil gone)		
Dec. 16, '22	20/20	20/20
Jan. 15, '24	20/20–3	20/20
Aug. 7, '24	20/20–2	20/20

DR. MARTIN COHEN, New York City: There is one question which I wish to ask Dr. Greenwood; that is, what focal infection could produce cataract? This fact might be important for the experimental study of the causation of cataract.

In a combined paper with Dr. Isaac Levin entitled "The Action of Radium on Cataracts," which was presented to the Section of Ophthalmology of the A. M. A. in June, 1919, we made the following statement: "That after one year's study of this subject, the lapse of time was too short to determine whether the initial retrogression of the lenticular opacities can be maintained under radium treatment, or whether sooner or later the radiation will cease to be effective, and the cataract go on to maturation."

We reported at that meeting the effect of radium on twenty-four patients afflicted with incipient cataract, who received the application at intervals from two months to one year, and stated that slight improvement occurred in the vision and opacification in 87.5% of the cases; that no ill effects to the structures of the eye were noted in any of the cases; that radium does produce some, if ever so slight, biologic alterations in the lens. It was therefore imperative that the clinical investigations be continued by competent investigators, their results checked and a definite conclusion reached.

Following this report, we practically discontinued the application of radium in these cases, but I still continued to observe and make notes on the vision and lens opacification at longer intervals for the past five years. I have operated upon fourteen cases following lens maturity with satisfactory results.

After five years of observation of the twenty-four cases of incipient cataract, fourteen progressed to maturity after discontinuing the application of radium from four months to one year; in six cases, the vision decreased and the lens opacification increased, but did not become entirely matured; in three cases, the vision and lens opacification remained practically stationary; in one case, the patient could not be followed up. The three cases where the lens opacification remained practically stationary for five years, occurs rarely; it even lasts, as is known, for longer periods, regardless of any treatment.

Whether the discontinuation of the application of radium after one years study was too short a period to warrant better results, I am unable to say. Still, the progress of the opacification of the lens in a few of the cases treated before we completely discontinued its use, made me

dubious of the efficacy of radium. I therefore decided to discontinue this study and await the report of other investigators.

In September, 1920, Drs. Franklin and Cordes published a paper in the American Journal of Ophthalmology entitled "Radium in Cataract." They confirmed our original report, and stated that improvement occurred in eighty-three per cent of their cases of incipient cataract.

In August, 1924, Drs. McKee and Sweet published an article in the American Journal of Ophthalmology entitled, "Use of Radium in Cataract." Their conclusion, however, was that radium as a therapeutic agent in the treatment of incipient cataract has very little value. This statement coincides with the results of my final observation in a way, except that in my case the final therapeutic effect on the incipient lenticular opacities was probably of no value.

With respect to the use of radium and X-rays producing ill effects on the structures of the eye, the animal experimentation by competent authorities do not agree in their reports, namely, by von Hippel, Birch-Hirschfeld, Chalupecky, Flemming, Tribondeau with Lafargue, and recently Northrop, Bagg and others.

Birch-Hirschfeld was able to produce ill effects upon the cornea, lens and retina of rabbits with radium, but Chalupecky's experiments gave negative results, and he claimed Birch-Hirschfeld's findings were due to improper technic. Bagg was able to produce cataract in young cats by the application of gamma rays of radium to the occiput. Tribondeau observed that the lens in new born animals was exceedingly sensitive to the X-rays, an exposure of five minutes being sufficient to produce cataract. In adult animals the reverse was proven.

Most authorities claim that the normal structures of the human eyeball are not sensitive to radium and the Roentgen rays when properly applied. Still, cases have been reported in the literature, where patients have been treated for lesion of the lid and eyeball, which produced a marked keratitis and cataract.

In August, 1924, an article by Dr. Fred M. Johnson appeared in the American Journal of Ophthalmology, in which he reports a case of a papillary growth of the eyeball in a female, age 25, who was treated with filtered radium tubes, resulting in a cataract formation.

Ten months ago, a male, age 63, was referred to our eye clinic from the radium department, with a history of having been treated with radium tubes for one year at the Post Graduate Hospital, for a basal cell carcinoma of the left lower lid. The right eye was normal in every respect; the left eye showed a large ulcerating tumor involving the entire lower lid, which extended into the orbit and invaded the eyeball, encroaching on the limbus. The vision equalled light perception; the projection was normal; the ocular conjunctiva was chemotic; the cornea was transparent, except over a small area near the limbus; the anterior chamber was shallow; the lens was cloudy; the intraocular tension was increased, and the patient complained of intense ocular pain. Exenteration of the orbit was advised and accepted. A brief report of the gross and microscopic examination is as follows:

The specimen measured 27 mm. anteroposteriorly, 28 mm. in the transverse diameter, and 24 mm. in the vertical diameter. The cornea

was transparent. In the horizontal plane, the growth extended to within 1 mm. of the limbus and 1 mm. of the optic nerve.

Microscopically, the corneal epithelium showed slight cornification of its superficial layers. The stroma was normal except near the limbus. The tumor cells on the eyeball were of the basal cell type, as found in the primary lid tumor. The iris showed mild infiltration, the choroid was congested, and the retina showed a wavy detachment and edema. The papilla was markedly edematous; the intervaginal space was dilated. In the gross, the lens was yellowish white and semiopaque. Sections of the lens showed no demonstrable structure with hematoxylin and eosin; that is, the capsule and epithelium were equally homogenous, suggesting necrosis. The lens substance throughout was practically the same. The condition of the lens was possibly due to radiation.

The effect of radiation of radium, X-ray and electricity on lenticular opacities has practically not been beneficial, but under certain circumstances deleterious.

These rays consist either of short or long wave lengths, and are either absorbed by the eye structures, when they act as caustics, or else can penetrate the structures without causing any ill results, but sometimes deep effects.

The dosage of the required rays can be determined, but it is practically impossible to calculate their effect solely on the deeper structures, except possibly with the X-rays.

It is known that in the ocular media, especially the lens, many of the ultraviolet rays are absorbed. In youth, according to Braun of Petrograd, very few rays of less than 376 millimicrons are absorbed by the lens. Wave lengths under 320 millimicrons are largely absorbed by the cornea. Rays between 350 and 400 millimicrons exert most activity on the lens.

The electric arc light contains rays of less than 300 millimicrons, which are absorbed by the cornea and conjunctiva, causing inflammation in four to six hours after exposure, indicating the effect of ultraviolet rays, while the ultrared rays produce instantaneous inflammation.

The absence of any permanent beneficial effect upon the lens with radium in our reported cases was probably partially due to the precautions necessary for protecting the vital structures of the eyeball with a brass plate and other filters, thereby producing no effect on the lens opacity, except possibly in the few cases where some of the deep gamma rays might have been absorbed by the lens, causing the temporary beneficial effect.

Continued experimental work, with aid of the slit lamp, is necessary for further study of the effects of radiation on the lens and other structures of the eyeball.

Dr. E. L. Jones, Cumberland, Md.: I am glad that Dr. Greenwood has brought up this subject, because I think for many years it has been a reproach to our profession that so many of these people are being permitted to go blind. I heard Colonel Smith, when he was here last time, speaking in Boston privately, say that there were thousands of people in New England who were going blind, who should not be permitted to go blind; and if they were given a certain treatment most of them would be saved. He said the uvea controlled the lens, and if it is given good

drainage, it will improve the nutrition of the lens. I have observed these things for seventeen years, since I began the use of nonoperative treatment of cataract in the early stages. I waited four or five years before I made any publication, but I have here a reprint of two papers, one of which is entitled, "Usefulness of Dionin in Early Senile Cataract," which was published in the Annals of Ophthalmology in October, 1913. The second is "Arrest of Cataract at an Early Stage," which was read in Denver, in 1915. What I want to say is embodied in these papers. Of course we know that cataract *sometimes* progresses so far and stops; but we know that it *usually* happens if you begin treatment early, and I have emphasized in this paper the fact that it is the early treatment. Cases such as thrown on the screen by Dr. Dean, show that beautifully. I have had several hundred cases in which I have used this treatment, and in the majority of these cases the patients have good vision. I have many of these patients living now, who started on this treatment fifteen years ago, and I have been able to see a great many of them from time to time and the vision is holding beautifully.

Since the program appeared, an old lady, now 78 years old, came in to have her eyes retested. She was tested once or twice in previous years, and then she was observed to be getting cataracts—that was in 1908. She had numerous striae, and the vision had dropped down to 20/60. She was given the dionin treatment and still holds her vision. In the last ten years, I have given my cases, in addition to the dionin, mercury with chalk and iron, which stimulates the liver and combats intestinal autointoxication and other things like that, which tend to produce a low grade uveitis, maybe unrecognized.

Dr. A. E. Davis, New York City: Lack of knowledge as to the exact etiology of cataract has been the chief stumbling block in the way of therapeutic measures used for its prevention and cure. Colonel Kirkpatrick, our distinguished guest of honor, and J. Burdon Cooper, have in recent years added much to the solution of the question of etiology. Roemer previously had advanced the theory that cataract was a specific metabolic disease. If we accept his views, and keep in mind Kirkpatrick's suggestion as to the influence of endocrin disturbance, we can arrive at a rational method of procedure in the prevention and absorption of lenticular opacities. Every patient who is found to have incipient or immature cataract should be subjected to the most rigid physical examination, together with laboratory tests, as to urine, blood, and so forth, and a Wassermann test made if there is any indication of lues. After a careful physical examination, a thorough local examination of the eye is in order, to note the nature of the opacities, with drawings, and where possible the slit lamp should be used. The vision should be noted and refractive errors carefully recorded.

Treatment directed to the correction of faulty metabolism and lessening the toxins which cause the opacities is of first importance, hence the necessity for correct diet, proper hygienic surroundings, exercise, and so forth, and the frequent and careful correction of refractive errors.

The benefits derived from local applications or injections for increasing the circulation of the eye, thus stimulating the metabolic processes within the eye, can be explained on the metabolic theory of cataract.

Such applications, alone, have given excellent results in incipient cataract, according to reliable authority.

I have had no experience with the use of X-ray, radium or electricity in the treatment of cataract, but here again, in my opinion, the improvement in the eye condition under such treatment may be explained on the assumption of increased metabolic processes within the eye itself.

The use of lens antigen as practiced by myself, assuming, with Roemer, that cataract is a specific metabolic disease, has a most important bearing on the treatment of cataract. In my opinion, it does not act as an ordinary protein, but as a specific protein, and has a selective action on the cataractous lens. Its action in the arrest and the absorption of the lenticular opacities can be logically explained in this way. In my hands, the results have been most gratifying. With the aid of general treatment and local irritative application to the eyes, the results should be even better than with the specific lens antigen treatment alone.

In concluding my remarks, I can but voice the words of Colonel Henry Smith, of cataract fame: "The question of cataract opens up a wide field for physiologists. I hope that physiologists will devote more attention to the nutrition of the lens and vitreous than they have done in the past." In other words, prevention is the main objective. In the whole range of ailments of the human body, I know of none of more importance than the arrest of cataract, which, if taken in time by modern methods of treatment, can be accomplished, with retention of useful vision in the great majority of patients.

Dr. Robert Scott Lamb, Washington, D. C.: I think, when one contrasts the two pictures of the patient, he will take a serious interest in this paper of Doctor Greenwood's—a patient losing his vocation or his pleasurable daily activities and being led around, or a patient waiting until such time as the lens may mature and be operable, according to the ideas of many. Anything that can be done to continue the patient's pleasure in life and his profitable employment, should be done. Therefore, I heartily agree with what Doctor Greenwood has said.

I think we must keep in mind that concomitantly with the opacity of the lens there is more or less cloudiness of vitreous, which the methods of Doctor Greenwood, or those of Doctor Dean and Doctor Jones, are equally efficacious in clearing up, and I think much of the improvement in the vision comes from improvement in the vitreous.

As to the point of selection, for injection of cyanid, I think we should avoid the upper outer quadrant, for the reason that it is in close proximity to the lacrimal gland and causes considerable disturbance and discomfort to the patient, whereas the lower quadrant gives no difficulty at all.

Dr. A. E. Bulson, Jr., Fort Wayne, Indiana: In my judgment, it is a mistake not to tell a patient when incipient cataract is discovered, but the information can be conveyed in a way that does not cause alarm. If we neglect to do this and later the patient discovers, through other sources, that an incipient cataract is present, we probably will be charged with having been careless in examination or wilfully dishonest. Furthermore, I am inclined to believe that patients will follow more carefully the much needed directions concerning attention to the general health and care of the eyes, if they know that hastening of the opacity

of the lens may be brought about through their own indiscretions. Then again, the patient also should be told that progress of opacity sometimes is exceedingly slow, and sometimes never gets to the point where it causes very serious impairment to useful vision, and especially if the patient follows the directions that are laid down.

I believe that treatment of incipient cataract is justified, and aside from attention to general health and advice as to the use of the eyes, I have been employing in these cases subconjunctival injections of dionin from two to three times per month, and quite recently have supplemented this treatment by the intravenous use of sodium iodid, in the belief that it aided in producing absorption and in stimulating nutrition of the lens. In some of my cases, there has been a clearing of the lens, and others apparently were unimpaired and went on to operation. In one case, both eyes showed striae shooting in from the circumference of the lens, and the treatment mentioned produced within one year almost complete clearing of the lens in one eye, whereas the other one went on to the development of a complete opacity of the lens and now is ready for operation. Therefore, I am firmly of the belief, as Dr. Greenwood has pointed out, that we ought to give these patients with incipient cataract more attention. Certainly, there will be a sufficient proportion of good results to justify us in following the recommendation that has been made.

Dr. David W. Wells, Boston: Dr. Greenwood and Dr. Bulson have both mentioned the fact that frequently cataracts clear up without any treatment. That brings to mind an idea which I have had for a number of years, that before we could judge as to the correctness of any method of treating cataract, we should have a tabulation of the natural history of untreated cataract. To that end, five years ago I sent around 1,200 reply postals, asking cooperation in accumulating data on the natural history of untreated cataract. I received about fifty affirmative replies, and sent to these men printed and ruled sheets for recording the results in cases where the vision was, at the time of the discovery of cataract, 20/30, 20/50, 20/70 and 20/200. Unfortunately, this work requires a good deal of time, and out of the fifty, I received only five or six reports, the number of cases being insufficient for any trustworthy generalization.

If we are to arrive at the exact facts regarding the treatment of cataract, is it not important to know what happens in a case of untreated cataract? And if it is in order at the close of this session, I will make a motion that a resolution be passed referring to the council the question of appointing a committee to obtain absolute and definite data about the natural history of untreated cataract.

Dr. Allen Greenwood, Boston (closing): I have nothing special to add to what has been said, except to call Dr. Bulson's attention to the fact, that while I dislike to talk cataract in incipient cases, I always inform my patients that there is some opacity in the lens which, if untreated, may result in mature cataract at some time, avoiding, however, as far as possible, a statement to the patient that he has a beginning cataract.

In regard to the question of Dr. Cohen, I referred to cases of cataract which apparently come from focal infection, to those cases where there is a definite but slight uveal involvement, and in such the removal of the infection causing the uveal disturbance would improve the patient's lens opacities.

BACTERIOLOGIC EXAMINATIONS OF THE CONJUNCTIVAL SAC AS A ROUTINE MEASURE IN THE PREOPERATIVE TECHNIC.

W. Gordon M. Byers, M.D., and A. A. Bruére, M.D.

MONTREAL.

In response to the kind invitation extended to us through your President, we have been glad to prepare the following statement on bacteriologic examinations of the conjunctival sac as a routine measure in the preoperative technic. The subject is one concerning the value of which there is a marked divergence of opinion; but it will be our task to consider the different points of view, and to try to reach a decision in regard to what is the right course to follow. At the outset, it will be necessary to set down a few facts by way of introduction.

The investigations of many workers have shown us that, apart from saprophytic bacteria, which are found in practically every instance, pathogenic organisms are present in a considerable percentage of patients, even with normal conjunctivae. It is not necessary here to fix the precise percentage in which they occur, nor are we concerned for the moment with the question of where pathogenicity begins and ends. Our purpose is served by the statement, that menacing organisms are frequently present, and that experience has shown that these microorganisms can give rise to destructive inflammations following operations upon the eyeball.

After a really vast amount of effort, we have to admit that sterilization of the conjunctiva cannot be achieved. Surgery has failed even with the skin, which is markedly less sensitive to chemical agents than is the conjunctiva. Occasionally, with measures that are known to all, an apparently germ free conjunctiva can be obtained; but it is a common experience to find the original bacteria again after an operation has been performed. The explanation of this fact is to be found in the difficulty of collecting the organisms, owing

to the numerous folds and pockets of the conjunctiva, the wide distribution and often scant numbers of the bacteria, and, as recently pointed out by Howard, the tenacity with which the germs adhere, not only to the conjunctival surface, but to the corneal surface as well. Indeed, the bacteria actually may be included in the epithelial cells. In no case, then, can we be certain of a sterile field in ophthalmic operations.

Fortunately, I think we can say without entering into the reasons why, the parts of the eyeball commonly involved in operations are relatively resistant to infection. This fact has been generally accepted since before antiseptic days. There is even accurate work which appears to show that in reality the results following operation are equally as good in those cases in which pathogenic bacteria are found, as in those in which the bacteriologic findings are negative. On the other hand, it is known that the virulence of a given pathogenic organism is by no means fixed. Even the pneumococcus, which stands at the head of the microorganisms producing intraocular infections, has a saprophytic phase, developing pathogenicity when conditions, such as one finds in an obstructed tear sac, are especially favorable to its development.

At the head of all the causes of infection stand imperfect sterilization, tear sac troubles, and inflammatory conditions of the conjunctiva. These sources are too well understood to call for further comment at this time; but other factors are not so fully appreciated.

Lack of operative skill is unquestionably one that must be taken into account. It lowers the vitality of the parts through undue traumatism; and it is responsible for avenues of entrance for the bacteria, through the creation of incisions that do not perfectly coapt, and through leaving behind debris and shreds of vitreous and iris that block the lips of the wound. In the same environment, the infections in the cases of an experienced operator are strikingly fewer than those that occur in the patients of less skilled men. An equally important factor is a faulty technic that permits of the entrance of organisms into the wound during the operation, as the result of one of the many "breaks" that are commonly seen.

The influence of the general health upon infections may be a debatable question, but the role of endogenous infections

in producing postoperative inflammations, especially of the plastic sort, is one that has come to be clearly recognized.

Perfect coaptation of the lips of a wound is a great safeguard against infection, but the conjunctival flap forms our most effective barrier to the entrance of bacteria into the eye following operations. Perfectly made, we can say with a great deal of assurance that it gives absolute protection.

Returning now to our subject, we can say that there are two main schools of thought in regard to bacteriologic examinations as a routine measure in preoperative technic. The one school holds that they are not essential or expedient, while the other feels that they are of value and should be carried out.

The first school is of the mind that routine bacteriologic examinations of the conjunctival sac are not justified by the value of the information they yield, since sterilization cannot be achieved, and all that one can do in any case is to fully meet the exigencies set forth in the preceding paragraph on sources of infection. It maintains, moreover, that routine bacteriologic examinations entail the expenditure of a great deal of money and time, and that they are a source of trying delays for the patient. They hold, too, that, in centers where the operative material is large, the measure is not practicable; while in the case of a large group of men, removed from a laboratory, and without either the knowledge or the resources to do the work, it virtually means that, if we insist on routine bacteriologic examinations, a great many surgeons must turn over their operative material to men who possess the bacteriologic facilities.

But, to be logical, the first school must be infallible in its selection of cases. It is notorious, however, that men's conceptions of what constitutes a healthy conjunctiva vary greatly, and that it is impossible by inspection alone to say whether a field is or is not contaminated. A smooth, glistening conjunctiva, free from congestion, may, as we have seen, harbor virulent microorganisms; while, on the other hand, a congested and roughened conjunctiva, with even a certain amount of secretion, may give only growths of harmless saprophytes. The ophthalmologist can as little afford to do without bacteriologic examinations in this field, if complete information is desired, as the surgeon can forego microscopic sections in determining what course to pursue with suspicious growths.

To be consistent, also, in the operative field, the adherents of the first school must secure complete protection, or at least coaptation, of the wound in every instance. No surgeon operating in the usual way can, however, invariably achieve this result. Even with the ordinary conjunctival flap, the incision is not always satisfying above, and it is frequently inadequate at the sides, where, in our experience, infection frequently takes place. To secure complete protection, most men would feel it necessary to have recourse to special flaps or sutures, but these, in adding to the difficulties of the incision, in causing hemorrhage and complications, and in prolonging the manipulation, may lessen our chances of a perfect result, in the way indicated in the paragraphs on infection. One must choose here, however, either measures of this kind, or adopt bacteriologic findings as a guide; operating in the ordinary way when the results are negative, and adopting extraordinary precautions when pathogenic organisms are found. In making a decision on this point, practicability and operative skill are important considerations.

From the scientific standpoint, there can be no doubt about the desirability of carrying on routine bacteriologic examinations; for without them there will always arise a gap between cause and effect. There are really very few thorough studies of panophthalmitis in its early stages, and our knowledge of the rôle of bacteria in relation to postoperative plastic inflammations is practically nil. Studies of large series of cases showing relative results in pathogenic and nonpathogenic cases are greatly wanted. The work of Lindner and of Howard has opened up a whole new field of investigation.

Summarizing, we feel we must conclude that routine bacteriologic examinations are essential to the scientific development of ophthalmology; that they are a necessary aid in determining the state of the field of operation; that they are our most rational guide to operative interference; and that they should be omitted only under stress of necessity.

But, if we adopt bacteriologic examinations as a routine measure, we must do so only with certain provisos. One is that the work must be knowingly and meticulously done, for imperfect findings are as dangerous as no findings at all. In this connection we would emphasize only two points: We are in entire accord with those who hold that the information yielded by smear preparations is entirely inadequate; and we

feel that the "liquid" method of collecting material from the conjunctival sac gives more accurate results than either the platinum loop or dry swabs. Our practice is to instill into the eye a few drops of water of condensation of Löffler's serum, using a sterilized nipple pipette for the purpose. By aspiration and expulsion of the fluid, the conjunctival surface can be well irrigated, and the washings thus obtained planted in the media selected.

Our second proviso is, that bacteriologic examinations must be regarded as ancillary only; for, if there is a danger in omitting them, there is also the danger that, in relying upon them too fully, we may neglect the indispensable routine inspection. In combining our clinical work with the work of the laboratory, we will best serve our patients and achieve our greatest measure of success.

DISCUSSION.

Dr. Harold Gifford, Omaha, Nebraska: The question of the methods by which bacteriologic investigation of the sac is to be carried out, is certainly of great importance. There can be no question that absolute sterilization of the sac is a practical impossibility. Even the last communication from Elschnig's clinic admits this. But the question is whether there can be a practical sterilization, and whether it is worth while to do it. I have reached the conclusion that where the conjunctiva and lacrimal apparatus are normal, a preliminary bacteriologic examination of the sac does not pay. In such cases, if the history of the patient is good, and if there is no other contraindication, all that I do is to send the patient to the hospital the day before, have him get chlorid of zinc, ⅛ of 1 per cent, and 10 per cent argyrol three times a day; and operate the next afternoon.

In my last 500 cases of senile cataract expression, I have had no suppuration of the cornea or any infection of the wound. On the other hand, in this list I had three cases where the sight was lost from low grade iridocyclitis occurring some time after a secondary discission. These results compare favorably with those obtained by the most careful preoperative examinations and precautions. I think a safe assumption is that the conjunctival sac is always infected, and that, beside doing the operation with as little traumatism as possible, one should wipe the surface along the line of the proposed incision with a moist swab immediately before using the knife.

What are we going to do about focal infection? Should we submit all our patients to thorough examination for every possible sort of focal infection, and should we insist upon such a source being eliminated if possible? This centers usually in old people around the condition of the teeth. Of course, sometimes you find bad tonsils or sinuses, but they are not apt to cause trouble in old people, although they should be investigated. But you very frequently find bad teeth in these old patients, and if you insist upon having their teeth fixed up or extracted

in all cases before you operate, you will have a good many of these patients leave you. In other words, you will find with lots of these old people, that if you ask them whether they will have the teeth extracted or take the risk, they will take the risk. When I think of the hundreds of cases I have operated without paying the slightest attention to the teeth, I am bound to say to these patients that the condition of the teeth will probably make no difference in the outcome of the operation. At the same time, I advise them that if I were in their place I would have a radiograph taken of the teeth, and if this shows any signs of root pathology, I would have the bad teeth taken out.

As to the danger from tonsils and sinuses in a person who has not been subject to tonsillitis, if he is old and has senile cataract, I think you can leave him alone, even if you do find a few crypts containing pus; but if he has a purulent sinusitis, this should be cleared up.

As to methods of investigation, the wire loop is notoriously poor for this purpose. But if you will take a flat spatula, the shape of a small razor, and scrape the surface, you will get astonishing results. This is shown by the fact that statistics on the xerosis bacillus run along, with most observers, from 25 to 50 per cent. If you scrape the surface and use the right kind of serum, namely, a serum which is solidified only to the point where it is still soft on the surface and nearly transparent, you will get the xerosis bacillus in practically 100 per cent of normal cases and the staphylococcus in from 75 to 90 per cent.

What shall you do to diseased tear passages? Shall you do an extirpation or destruction of the sac, or one of the modern operations for obtaining a new opening into the nose? It seems to me that it is a dangerous thing before a cataract operation to open up the conjunctival sac to direct communication with the nasal passages. I believe, so far as the cataract operation is concerned, it is safer to extirpate or destroy the sac. Thiel says he finds the number of pneumococci in the conjunctival sac after a successful Toti operation to be decidedly increased. My advice is to destroy the canaliculi as well as the sac. We had one case of serpent ulcer in which the sac was destroyed, with a perfect result on both sides. There was no pus or discharge, and yet this patient, who lost one eye from the first ulcer, came back several years later with a pneumococcus ulcer on the other eye and nearly lost that, probably because we had failed to destroy the canaliculi. So you should open the canaliculi down to the upper part of the sac, and then destroy all the membrane and the surrounding tissue by the vigorous use of the cautery.

Dr. Harry S. Gradle, Chicago: It is particularly gratifying to hear the statement made by Dr. Byers regarding the routine bacteriologic examinations of the sac, because that is the school I was brought up in.

In the original investigations of Elschnig, he showed practically 46 per cent of normal appearing conjunctiva contained streptococci, another equally large per cent, staphylococci, and these figures have been corroborated by recent investigations. Wissman reported streptococci found in 42 per cent of all cases of senile cataract. Stanka, in 401 eyes, reported pneumococci, streptococci and staphylococci in three tenths of all cases of senile cataract. Why do we not obtain an intraocular infection at operation more frequently than we do, in view of the fact that practically all eyes are infected? There are several reasons. In the first place, we must

differentiate between the organisms that lie upon the free conjunctival surface, and the organisms that lie within the superficial or the epithelial cells. The latter, I believe, play practically no role in the formation of intraocular infection; the former are apt to be of low virulence, and unless particularly favorable circumstances arise, which will allow increase in virulence, they seldom cause trouble. From the time the operation is performed until agglutination takes place is not more than three or four hours. Agglutination and closure occur rapidly, and all preparation and asepsis of the conjunctival sac is aimed at the first few hours.

A large laboratory is not essential for routine bacteriologic examination of the conjunctival sac. It can be performed by anybody with a microscope. A smear is of no value. In a series of cases examined in 1911, I showed that only 60 per cent of cases in which organisms were found on culture, showed organisms in the smear. Therefore, fluid culture is essential to determine the presence of organisms in the conjunctiva. A good culture medium is one part normal horse serum and two parts bouillon. The normal horse serum can be obtained from any large pharmaceutic house. The bouillon is a simple preparation. This must be mixed immediately before preparing the culture. Mixing the two and allowing them to stand will render the culture valueless.

Material from the conjunctival sac may be obtained by the pipette suction method, in which normal salt solution is instilled into the sac and drawn out; or by the Cone method, in which a sterile silk thread is dropped into the conjunctival sac, allowed to remain a few moments, and then removed and dropped into the culture tube. After determining whether or not the conjunctival sac is sterile, it behooves us to endeavor to clean the sac as far as lies within our power, in case of the presence of pneumococci, streptococci, or the large or small staphylococci—not because we necessarily fear an infection, but it is a precaution which must be taken, and we are not justified in overlooking it. Examinations by Stanka of a large number of cases seem to show, that the most rapid cleansing of the conjunctival sac to as near the point of sterilization as possible, is accomplished by the frequent washing of the conjunctival sac with a 1 to 5,000 oxycyanid solution. This is not irritating, and can be carried out by the patient at home. It is further increased in rapidity by the use at night of the socalled Betti's salve—a 2 per cent yellow oxid of mercury ointment. In the case of the normal appearing conjunctiva, if these organisms are occasionally found present, we may be sure we can clear the sac sufficiently for ordinary operative procedure in five days. Where the sac is infected and the margins of the lids are infected, and where the tear passages are stenosed, we have an entirely different matter, but that should not be considered under this heading.

In conclusion, let me second what Dr. Byers suggests, that we carry out the examination of the conjunctival sac by the cultural method alone as a routine measure before cataract extraction, as a safeguard against possible future trouble, a panophthalmitis, an endophthalmitis, or a low grade of iridocyclitis that frequently follows cataract extraction.

Lt. Col. Henry Kirkpatrick, London: I am in thorough agreement with Dr. Byers. But while sound in theory, I am reactionary in practice. I confess I do not always insist upon a bacteriologic examination. The reason is, that in a large hospital practice, such as we have in the East,

it was impossible. But we did this—we first of all excluded any patient with an obvious discharge, or any obviously unhealthy condition of the conjunctiva, and put him aside for further treatment. We found the use of Herbert's method of disinfection of the conjunctival sac exceedingly valuable. There you know the fornices are inverted forcibly, and a stream of chlorid of mercury is poured over the eye. This excites considerable reaction and a superficial necrosis of the conjunctival cells. The patient is brought to the operating table with a hyperemic conjunctiva. A certain amount of this particular necrosis is the result of the treatment. This mucopus we used to consider was composed of degenerative cells, and the bacteria on the surface of the conjunctiva were caught in these cells as fish are caught in a net, and we went in and washed them out with normal saline. That was our practice, and we found that it worked to all intents and purposes. Since coming West, I have rather altered my practice, and now I depend upon silver salts of various kinds, and of these I value very highly one per cent nitrat of silver. I think, however, that the nitrat of silver applications should be stopped a few days before operation. I cannot help thinking it is a safeguard.

The resistance of the ocular tissues to infection has been mentioned. I think that is a surprising thing, and one marvels at it in the East. Frequently, we find patients who have had their lenses couched, or an attempt made to do so, where the accoucher has no idea of asepsis nor does he select his cases very carefully. Yet we get extraordinarily few cases of suppurating panophthalmitis. Of course, when I say few, I mean that of the lost eyes which one would see from couching, probably 15 per cent, at a rough guess, would have panophthalmitis, whereas the greater number would be due to secondary glaucoma.

Dr. George F. Keiper, Lafayette, Ind.: The question is whether we shall make a routine examination of the conjunctival sac for bacteria. By all means, yes! I want to emphasize the point that Dr. Gradle made, that a smear is not of much value; it must be a culture.

The question also has arisen as to the possibility of the sterilization of the conjunctival sac. Let us make every attempt possible to sterilize that sac, both locally and also through the general treatment of the patient. Dr. Gifford has suggested chlorid of zinc locally. I prefer sulphat of zinc, but no matter what it is, let us make an effort anyway. A number of years ago, when I began the practice of ophthalmology, somebody strenuously recommended that before any operation for entering the eye, we should prepare our patient by giving internally the following mixture: 1½ grains of bichlorid of mercury with 5 drams of iodid of potassium, 2 ounces each of syrup of sarsaparilla and water, giving the patient one teaspoonful of that every three or four hours for a week or so before the operation. At that time, it was considered to be an alterative to get the patient in better shape for operation. We know now that iodin is secreted by the mucous membrane of the body as well as by the skin itself, and I think it would be a mighty good thing to prepare patients that way in order that the iodin might go into the conjunctival sac.

SECONDARY CATARACT OPENING BY SINGLE STRAIGHT INCISION. IRIDOTOMY BY SAME METHOD.

JOHN M. WHEELER, M.D.

NEW YORK CITY.

Unfortunately, many surgeons of large experience consider division of after cataract difficult in the extreme. Dread of this operation has had an influence in leading some ophthalmologists toward the adoption of intracapsular cataract extraction. I think there is quite general agreement now, that the degree of security which the posterior lens capsule affords during cataract extraction is well worth while, and that capsulotomy extraction is the wise choice for most ophthalmologists. So suggestions for handling secondary cataract are not out of place. A suitable operation for division of a secondary membrane must offer a good chance for a permanent opening in the visual line, and it must offer the possibility of only very slight traumatism to the eye. It must be an operation which one can approach without dread, but with confidence that a satisfactory opening will result. Familiarity with a definite and effective technic is an important requisite, and it is my purpose to attempt to make clear a procedure which I have practiced for years, and which a surgeon can rely on to give a sufficient permanent opening in after cataract, offering to the eye the least possible trauma.

D. W. Hunter developed a clever technic for opening a secondary membrane, and with it won the admiration of all who saw him operate. He used a ground down Graefe knife, and got a long, clean opening by sliding the blade on the membrane in a single rapid act of supination of the forearm. The results were sure and beautiful, but fear of slashing the cornea kept most surgeons from attempting the trick, and only a few emulated him successfully. The method which I wish to advocate is unbelievably simple and safe, and it offers the same sort of results which Hunter obtained.

The Knife.

The surgeon should have something better than a worn down cataract knife for making an incision in after cataract. The blade should have a keen point and a faultless edge, and it should be accurately shaped. The instrument I use has a blade 18 mm. long and 1 mm. wide at its widest part. It is carefully tapered and sturdy enough so that it will not bend. The edge is ground sharp throughout the length of the blade. Figure 1 illustrates the knife.

Technic.

Let us assume that there is a coloboma above from iridectomy, and that the pupil is dilated. The technic must be modified if only a short incision is wanted. The surgeon stands at the head of the table and a little to the left of the eye to be operated on. The speculum is introduced, and the eye is fixed by grasping the conjunctiva and subconjunctival tissues below the cornea at a point diametrically opposed to the point where the knife is to enter the cornea. The fixation forceps is held in the left hand without the slighest pressure on the eyeball. The handle of the knife is taken most delicately between the pulps of the forefinger and thumb of the right hand 2 inches (50 mm.) from the knife point, and care is taken that there is no tendency for the finger to wrap itself around the handle. This would render the execution of the technic impossible. The insert (c) in figure 2 is intended to show how the fingers are placed on the sides of the knife handle 2 inches from the point.

The eye looks slightly downward, and the point of the knife enters the upper part of the cornea 1 mm. from the limbus, in front of the coloboma. The incision should be planned so as to go through the heaviest bands if possible, but the bands can be cut at any angle; and it is well to carry the incision into the coloboma, so that the incision in the membrane will not be limited by the iris. (a) The knife point is carried deliberately across the aqueous chamber and behind the inferior portion of the iris, and carefully brought in contact with the membrane. One can take all the time he wants in this part of the procedure, provided the fixation forceps and knife are held in absolute relaxation. The rest of the operation is done speedily so that the incision is completed before the secondary membrane begins to relax from

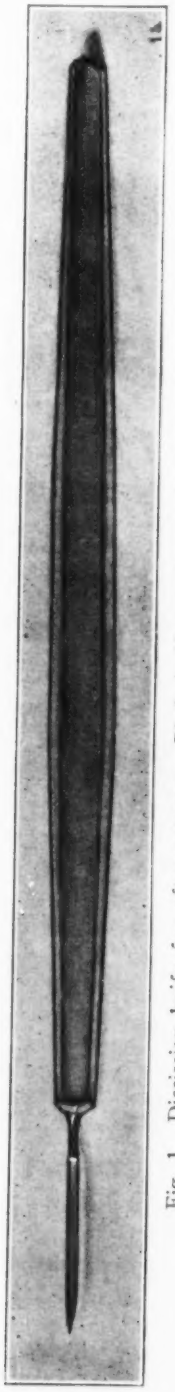


Fig. 1. Discission knife for after cataract. Blade is 18 mm. long and 1 mm. wide.

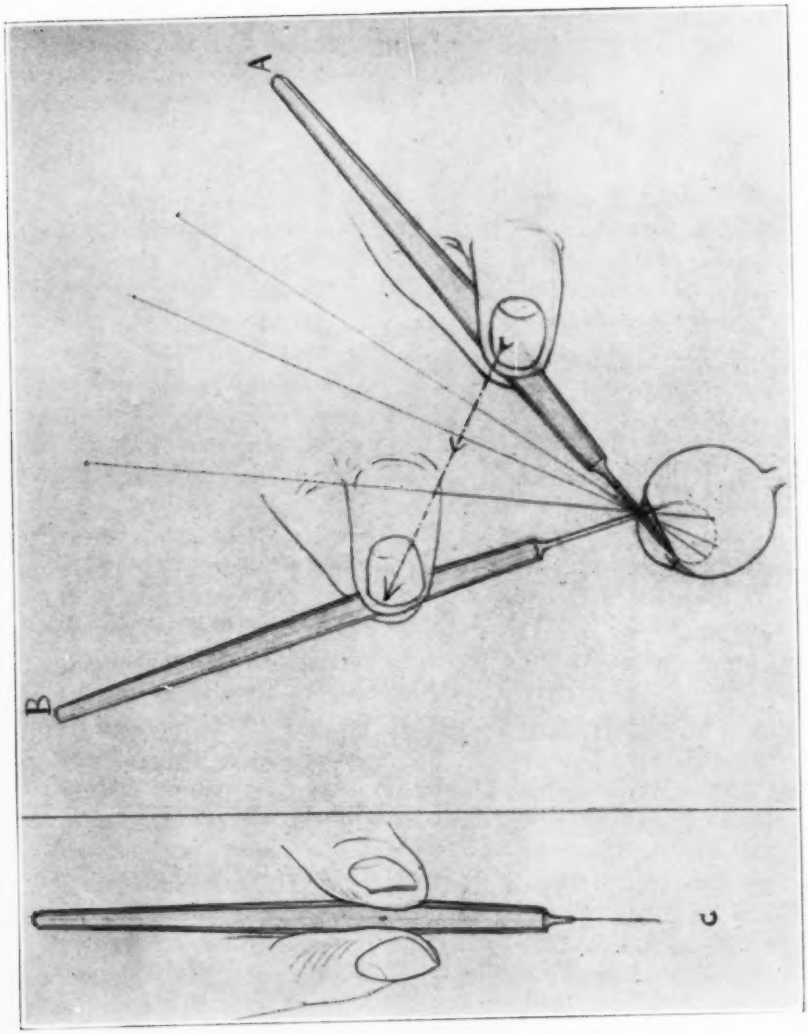


Fig. 2. Mechanics of discission. Finger and thumb hold knife lightly, and are carried along arrowed line. Knife point courses in vitreous body along dotted line. C gives view of back of knife as held in fingers.

division. By an absolutely simple movement on the part of the surgeon, the point and cutting edge of the knife blade are put through just the sort of an excursion that is needed to produce a long clean incision in the membrane without enlargement of the surface wound in the cornea. (b) From the position in which the knife is held with the point in gentle contact with the secondary membrane, the part of the knife handle between the pulps of the finger and thumb is carried in a straight line by a free rapid movement of the arm, without any change in position of the hand or wrist. By this movement the blade is made to slip in and out of the vitreous

Fig. 3. Opening in secondary membrane after discission. There is slight angulation of edges where a band in membrane was cut.

without any interruption in the sliding motion on the membrane, and the noncutting edge of the blade receives gentle pressure against the cornea until the blade slips out of the corneal wound, usually without loss of aqueous. Figure 2 shows the mechanics of the process. As the ends of the forefinger and thumb are carried along a straight line from position "A" to position "B," the knife handle changes position as though working freely on a pin passing straight through the knife handle and set in the pulps of the forefinger and thumb. This imaginary pin passes straight along the course indicated by the arrowed line in figure 2, and the knife slides first in and then out as it goes across the eye, and the operation is done. The insult to the eye has been almost none.

The result of this act is a straight incision which gradually opens up and never has a tendency to close. Manifestly, the opening must be in the visual line, but the width is not important as long as it is properly placed. Figure 3 shows a typical opening by this method. In this illustration, the effect of severing a band is shown by a slight angulation in the edges of the opening where the band was cut. There is no membrane, no matter how heavy the bands, that will not open freely by this method if the knife and the technic are correct.

Iridotomy.

Iridotomy can be accomplished by the same maneuver. The length of the incision is governed by the distance between the point of the knife and the place on the handle where the ends of the thumb and forefinger rest. The nearer to the knife point the fingers are placed, the shorter the incision in the iris will be. The scheme shown in figure 2 can be used to work out accurately the length of the membrane incision which will result from any finger position on the handle.

If the iris is drawn strongly toward the wound in an aphakic eye, an incision across the fibers, if of proper length and properly placed, will result in almost a round pupil.

Figure 4 shows a suitable eye for iridotomy, with a dotted line to indicate the incision in the iris. This eye suffered from recurrent attacks of iritis, and glaucoma had developed. Iridectomy had been performed, with injury to the lens. Absorption of the lens was allowed, and the condition shown in figure 4 ensued. Figure 5 shows the opening which resulted from the single horizontal incision, made by the same technic as outlined for after cataract, only the handle of the knife was held nearer the point. By this technic the iris and underlying membranes are easily cut.

Slit Lamp Corneal Microscope Observations.

The examinations were made with the cooperation of T. H. Johnson, whose superior skill was relied on. I had hoped that observations under high magnification would lead us to the conclusion that my method gives less damage to the eye than other methods, in which it would seem that prolonged manipulation might cause more disturbance to the vitreous than a quick single incision with a keen cutting instrument. But comparisons of findings in cases operated on

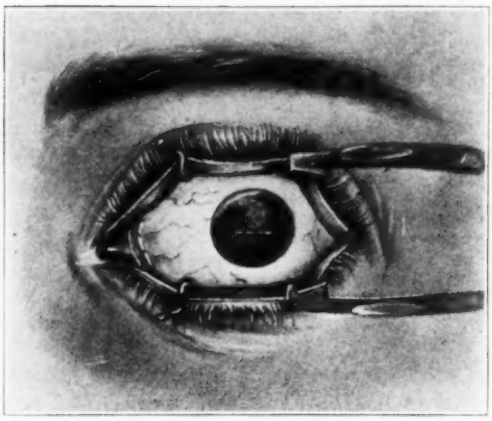


Fig. 4. Obliterated pupil in aphakic eye. Incision made along dotted line.

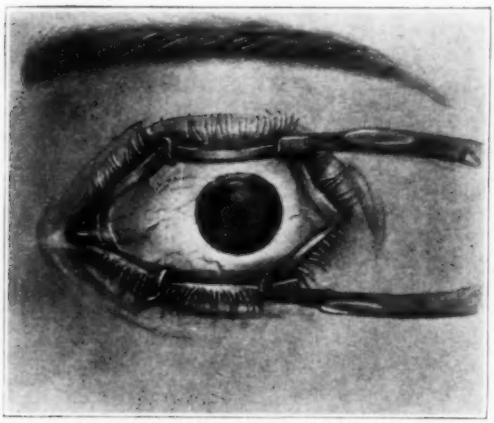


Fig. 5. Opening which resulted from single horizontal incision through stretched fibers of iris and underlying membranes.

by my technic with those operated on by different sorts of knife needles offered nothing convincing in regard to the degree of disturbance in the vitreous. In some eyes which received my maneuver, there was what appeared to be a definite vertical cleft in the anterior vitreous structure, which presumably resulted from the single incision, and the walls could be followed with the light bundle and the microscope. This opening might appear to be of advantage, but probably it offers no appreciable difference in favor of visual acuity. Moreover, there is a possibility that in some cases we were focusing between normal vertically disposed folds. In his atlas, Vogt shows in figure 334 what he calls "typical tunic like vertical folds," and in figure 339 "the step like antero-posteriorly arranged normal area of folds." But we think that we have not been confused by this striking arrangement of hanging folds which presents itself in the anterior part of some vitreous bodies observed under high magnifications.

After division of the membrane, vitreous enters the aqueous chamber, and the outline of the intruding vitreous and the particles of structural elements can be seen in detail. There are fibers, membranous particles, remnants of the fetal vascular system of the vitreous, greyish-white spots and pigmented dots. These tremble with movements of the eye, but do not float freely in the aqueous. It is not possible by any method to cut a secondary membrane without entering the vitreous, and, as the instrument is removed from the eye, the viscid vitreous has a tendency to stick to the instrument and follow it out of the eye. At the wound the vitreous is stripped off the blade and usually adheres to the wound, even if none follows out with the knife so as to present. So in most cases, after division of the after cataract by any method, the vitreous can be seen slanting forward toward the wound, and in some cases the attachment to the wound can be seen. Such attachment of vitreous to wound has to do with the development of iridocyclitis and glaucoma in a very small percentage of cases operated on, but in most cases is not of importance. The study of the vitreous body under high magnification is new business for the oculist, and our findings may assume new meanings after better acquaintance.

DISCUSSION.

Dr. Albert Lassalle, Montreal: I think we owe much to Dr. Wheeler for having brought before this meeting so interesting a subject. His new technic in the operation of secondary cataract I consider a step forward in our surgery.

Secondary cataract is very important in itself, and our patients are more or less impressed when, let us say, before the lens extraction, or sometimes afterwards we tell them that perhaps a second operation will be necessary.

It may sound rather strange to talk of secondary cataract in these days when oculists of every country are aiming at the extraction of the lens in its capsule, putting forward new methods which they say will surely prevent the formation of secondary cataract. Unfortunately, we have not yet attained to that high degree of perfection. We welcome any new technic for secondary cataract, and the method just described by Dr. Wheeler is worthy of our serious attention.

We all know the different aspects of the secondary cataract. First, there is that grayish scum formed by lenticular fibers on the capsule; sometimes the pupil is filled with white, snowy masses which may be absorbed or are persistent. There is another type of secondary cataract called latent secondary cataract, which may appear the second or third year, or even later. We can add to this a secondary cataract resulting from infection, operative complications, where we can see white connective tissue between the pupillary border and the cataract itself, which will not permit any dilatation by atropin.

As there is no actual means to prevent the appearance of secondary cataract, no matter what kind of operation has been performed, simple or combined extraction, or the extraction of the capsule itself, for in most of the cases this extraction is only partial and statistics gathered from everywhere amply prove this, we are perforce bound to look for a technic which will give the best permanent visual results with the least complication. The methods most commonly resorted to are:

1. The cystitome or needle. Very often the opening made by the incision is not large enough, or both sides of the capsule do not retract sufficiently to give the desired aperture.

2. The operation of a secondary cataract with a punch may give a good result if the secondary cataract is thick enough. With this instrument there is no pulling on the ciliary body.

3. The extraction by means of forceps (Kalt's forceps, for instance) gives a very good result if you happen to bring out the whole capsule, but most of the time this extraction is only partial. Using this method, one must be careful not to pull on the iris or ciliary body.

4. Of course, iridocapsulectomy will be resorted to when the secondary cataract is attached to the iris by conjunctival tissue, the sequel of a postoperative infection. In such cases, too much pressure on the iris would be dangerous, and the needling would not give any satisfactory results on account of the thickness of the capsule.

I think all these methods are more or less satisfactory, but extraction will be made only when the adhesions of the secondary cataract to the iris are very slight, and the ideal operation ought to be the cutting of the capsule with a von Graefe knife.

Surely we all agree that the ciliary body must be left alone; if this rule is not respected, trouble may arise in the future. I say it is quite impossible to extract totally a secondary cataract with the forceps, no matter what kind, without pulling more or less on the iris. You may object that sometimes only a part of the secondary cataract is extracted. That is true, but one can never tell before the operation whether the extraction is to be partial or total. Such a technic cannot be recommended, and furthermore, when you tear out a part of the capsule, you are not sure that it is always at the optimum point of vision. I think a Graefe knife, very sharp, is the best instrument—not an instrument that tears or pulls. With this knife, after transfixion, you can cut where you like at the chosen place, for the blade is long enough. You can know exactly before the operation where will be the aperture, its length, and its direction. One may object that the aperture created is likely to be covered later on by epithelial tissue or by the vitreous body being resorbed. But those who have been practicing this method for many years say that they never noticed any neoepithelial membrane, examinations having been made with the corneal microscope and the slit lamp. Certainly, you may have a recurrence of a secondary cataract. This will happen with any method, when inflammatory reaction sets in and transforms the internal layers of the vitreous body into an opaque membrane. The section with a knife applies to every kind of secondary cataract, thin or thick, comprising the capsule, masses, exudate, and the iris. The section is the more easy if the secondary cataract is hard and rigid.

All these advantages seem combined in the technic so ably described by Dr. Wheeler, and once more I thank him for having brought to our attention a modification of the operation for secondary cataract.

Dr. Arthur J. Bedell, Albany, N. Y.: Doctor Bedell showed many lantern slides illustrating the normal vitreous, the intact posterior lens capsule after operation, various types of secondary cataracts, and many forms of prolapse of vitreous following capsulotomy. He stated that Dr. Ziegler's V-shaped capsulotomy had served him so well that he had never performed the operation proposed by Dr. Wheeler.

He did not think it probable that the vitreous stuck to the knife as it was withdrawn from the wound, nor did he believe it wise to make a corneal incision when one protected by conjunctiva was the safer. An inexperienced operator might easily do much damage with the long knife of Dr. Wheeler, by forcing it too deeply into the vitreous or even striking the ciliary body. Any operation which causes intraocular hemorrhage produces more intraocular changes, with tendency to the formation of traction bands, complete pupillary occlusion, or even secondary glaucoma.

THE VALUE OF THE FUSION FACULTY AND METHODS FOR ITS DEVELOPMENT.

DAVID W. WELLS, M.D.

BOSTON, MASS.

Binocular single vision is a rather intricate psychic facutly, dependent on certain exact physical conditions.

The first physical essential is the semidecussation of the fibers of the optic nerves, and the second is the proper coordination of the 12 extrinsic muscles, so that the retinal images shall fall upon corresponding points of the two retinae.

When it is realized that a deviation of less than a millimeter in the position of corresponding points means diplopia for small objects, it is almost inconceivable that any mechanics alone could secure the desired result.

THE FUSION FACULTY.

According to Worth, sight in the newborn is limited to fixation of a light, so that all the finer qualities are the result of personal experience. The involuntary movements of the eyes of infants would show that there is no conception of binocular vision. The full development of the fusion faculty is not attained until the fifth or sixth year with the normal child.

Physiologic diplopia means that objects nearer or farther than the point fixed are always seen double, but the adult has so far succeeded in ignoring this, that it is sometimes difficult to make him realize the doubling.

There can be little doubt that this fact plays a very important rôle in the child's experience—the fusing at different distances developing the idea of perspective.

The argument is here introduced to emphasize the fact that the third essential to binocular single vision is the psychic one of the fusion faculty. Without such an overruling guidance, the necessarily exact coordination is inconceivable.

The occasional clinical experience, that loss of sight of one eye is frequently followed by divergence, is an unanswerable argument for the importance of the fusion faculty in

keeping eyes straight, and it plays quite as important a part in the production and the cure of those *tendencies* to turn, grouped under the name heterophoria.

The Third Dimension.

A person devoid of a fusion faculty, or with only one eye, learns by experience to calculate distance by rapidly fixing the eye on objects to one side, and thus establishes a parallax which enables him to measure the distance by a sort of muscle sense.

The inferiority of this sort of estimation of distance, compared with that possible to one with a well developed binocular fusion sense, was well shown by Howard at the Mineola experiment station. He claimed that many accidents in landing were due to the aviators' deficiency in estimation of depth difference.

Just how valuable this faculty is to chauffeurs, engineers and street car motormen has never been critically determined. When the facts are known, it is quite possible that visual tests will not stop with measuring the acuity of each eye and testing the color sense, but that a certain standard of stereopsis will be required. Four years ago, the writer[1] presented a simple plan for the routine office measurement of stereopsis or depth difference, which requires only five minutes.

In 1890, Prof. Wells, of the Massachusetts Institute of Technology, found that some students had great difficulty in comprehending the diagrams in solid geometry, and had stereoscopic charts made to overcome this deficiency. Several of these have been included in the author's selection of stereoscopic charts.

While a good fusion faculty is always a valuable possession, and in many walks of life absolutely essential, it is cheerfully admitted that one sees many patients totally lacking such a sense. These people use each eye separately and are not conscious of any loss, and have no asthenopia from incoordination.

Fusion faculty is also a matter of degree. Some patients are able to fuse concrete pictures by a rapid alternation of the two eyes, but have no conception of perspective. Other cases are able to fuse large concrete pictures and have some perspective, but fail to fuse small types or characters. Simultaneous macular perception is essential to true binocular fusion.

The author is familiar with Gould's[2] theory of dextro- and sinistroocularity, but has not found it to coincide with the facts. Undoubtedly, many people who habitually suppress one eye may not suffer any annoyance from this condition. The same may be said of errors of refraction, but does any one for that reason consider astigmia physiologic?

The writer maintains that the habitual use of the two eyes binocularly, with a minimum suppression of either, is as ideal as is the emmetropic eye to monocular vision.

Value of Training Fusion Faculty in Abnormal Conditions.

Esotropia: Every ophthalmologist is probably familiar with the pioneer work of Landolt, Javal and Worth. The question of whether the amblyopia causes the heterotropia, or the lack of the fusion faculty causes the heterotropia with an amblyopia ex anopsia will always bring out a lively discussion.

Worth has certainly presented strong evidence that the vision can be maintained if the fusion be developed, but all who have followed his methods have found plenty of cases in which the amblyopic eye was not materially improved.

Landolt[3] sums up the matter as follows: "Of course I have always considered the limitation of the temporal excursions of the eyes effected with convergent strabismus as the consequence and not the cause of strabismus. The abductors in this case lack exercise, especially in the symmetric movements. If, on the other hand, the absence of binocular vision is one of the principal causes of strabismus, the defective direction of the eye in turn becomes an obstacle to the development of this function. The individual thus finds himself in a vicious circle; he squints because his binocular vision is not developed, and binocular vision is not developed because he squints.

Tscherning[4] advises treatment under five headings:

"(a) Reestablishment of diplopia and, if possible, vision of strabismic eye.

(b) Reestablishment of the approximately correct position of the eyes by way of operation.

(c) Stereoscopic exercises.

(d) Exercises without the stereoscope.

(e) Controlled reading."

Certainly four of the above recommendations are fusion training.

In what proportion of cases esotropia can be cured by orthoptic means alone is difficult to answer, but it is certainly enough of a success so that in no case should one neglect to give it a good trial.

It is here that the amblyoscope of Worth is most useful, but great patience and intelligent parental cooperation are essential. Unfortuntely, one cannot watch the child's eyes, and is entirely dependent on his correct report of what he sees. We still fail to get the cases early enough.

The value of postoperative training of eyes cosmetically straight has been advocated by Landolt, Tscherning and Javal for many years, and it is to be regretted that it is almost universally neglected. The remaining heterophoria may be more annoying to the patient than his previous heterotropia.

Heterophoria: According to Hazens[5], Dr. E. Dyer, 1865, was one of the first to call attention to eyestrain from muscular imbalance. At the meeting of the American Ophthalmological Society, he read a paper entitled, "Asthenopia Not Connected With Hypermetropia."

Up to this time the teaching of Donders, that asthenopia (which we now call eyestrain) was always due to ciliary fatigue, was generally accepted. At the present time, many of us believe that heterophoria is almost as frequently the cause as errors of refraction. When the two conditions exist, it is often difficult to decide which is the more potent. The general rule seems to be to correct the refractive error, hoping thereby to relieve the heterophoria, but many times it is quite sufficient to correct the heterophoria.

This has usually been done by prescribing prisms either alone or incorporated in the refraction correction, but, except for vertical errors, the writer usually reserves this as a last resort. Tenotomy and advancement are indicated in selected cases, but a discussion of surgical indications is beyond the scope of this paper. Exercises with loose prisms both at the office and at home have been very generally *advocated* and *occasionally* practiced, but have not been attended with any general or lasting success.

Heterphoria is the field in which fusion training is most important. The results in esophoria have not been very encouraging, and less so in hyperphoria unless associated with exophoria. Exophoria, convergence insufficiency and pseudoesophoria are most amenable. By pseudoesophoria is meant

a condition in which the convergence duction is really weak, notwithstanding all tests except duction show an esophoria. In the course of developing a better convergence, a hyperphoria will often disappear, or such a tendency may be produced. Should it persist, it is corrected by means of a vertical prism combined with the lenses worn.

It has puzzled the writer that Worth, who has made such extravagant claims for fusion training in curing *heterotropia,* should make so little use of it in treating *heterophoria.*

Before treating the heterophoria, lenses are used for all they are worth, according to well established, orthodox principles, unless the refractive error is slight and the heterophoria seems to be the active cause of the eyestrain.

Javal, 1896, demonstrated the application of fusion training in heterophoria, which he calls latent strabismus.

Methods for Developing Fusion Faculty.

In discussing this subject in 1902, the writer made the following statement: "The rapid development of the adduction which is often obtained by socalled gymnastics, strongly suggests that the gain is not a muscle hypertrophy but an increase in innervation, either in the responsiveness of the end organ in the muscle or the convergence center or both. In the educational treatment of tabes, the incoordination is overcome by teaching the patient to gauge his motor impulses by the eye, in lieu of the normal sensory control. Repeated artificial contractions of the internal recti (the ciliares remaining relaxed) establishes a habit of increased action, so they no longer lag when the impulse to converge *and* accommodate is felt. The coordinating center may also be taught to better appreciate the advantages of binocular perspective.

This is no special pleading, but is analogous to other sensations. The pianist makes his fingers educate his brain, that the brain may do better work with the fingers. Tasks consciously performed are in time relegated to subconscious control. If this interpretation of muscle gymnastics be accepted, it is evident that the first indication is to teach the patient the fascination of true binocular fusion. All myology resolves itself into neurology, and behind the nerve impulse is a fusion faculty."

Twenty years' experience has not changed the writer's opinion of the main truth of this statement. Dr. Lancaster's

very scholarly exposition of the "All or Nothing Theory"[6] is an important confirmation. He is undoubtedly right in arguing against a fusion *center,* and in demanding in its place the conception of a fusion *faculty.* His idea, that the development of this faculty does not mean increased *strength* of nerve impulse, but the utilization of a greater number of unused nerve fibers, is a great help in understanding *how* it is done. But, whether it be a stronger impulse, or a greater number of impulses, that is the essential for establishing well worn pathways of motor discharge, it is still *more nerve impulse.*

Dr. Jackson says[7]: "Orthoptic exercises are for nerve training—all forms of orthoptic apparatus depend for their usefulness on the presence of a *fusion sense.* But whether this sense is strong or feeble, real exercise is possible only by the patient *making the effort.*"

Since it is the consensus of opinion that more nerve impulse is the first essential, and that this is absolutely dependent on a fusion sense, and that the patient must *make the effort,* it seems evident that success depends upon *cultivating* the *fusion faculty* by a *method* which *incites the patient to make the effort.*

For many years the writer has advocated stereoscopic training as the best means to this end. Many practitioners give to the patient a stereoscope and a set of cards, and send him off "to work out his own salvation." This is a good deal like expecting a man to learn trigonometry by giving him a text book. The average man needs teaching, supplemented by the home use of the stereoscope, the *cards selected* so *as to give him* a *definite lesson* to *learn.*

For office training the phorooptometer with two revolving prisms affords a simple means of making a stereoscope to order, to fit the patient's individual needs.

Since, in treating convergence insufficiency, it is desired to associate a greater convergence impulse with the accomodative impulse, the tendency to accommodate is restrained by using + 10 lenses and having the card holder fixed at 10 cm. distance. Any attempt to accommodate blurs the picture. The rotary prisms are adjusted so that the lines of sight cross at 6 cm., if one is using cards with corresponding points 6 cm. apart. Then concrete pictures are used, commencing with those of large enough characters to be easily fused, and progressing to the more difficult. As soon as the fused picture lacks any part of the two separate pictures, attention is called to the

failure and the patient is helped to overcome this suppression, by blurring the image of the dominant eye by an extra + lens inserted behind the + 10. Or he is taught to partly close the dominant eye, so that the light to that eye is filtered thru the lashes. As soon as small characters are easily fused, perspective cards are then added.

The cultivation of convergence requires *increasing the prismatic element of the stereoscope while the patient holds the image fused.* Naturally one would do this by turning the revolving prisms. Formerly, the half pictures were inserted in the clips, which by a right and left screw could be brought nearer together. Both of these methods were found to disturb fusion before much extra prism could be exhibited.

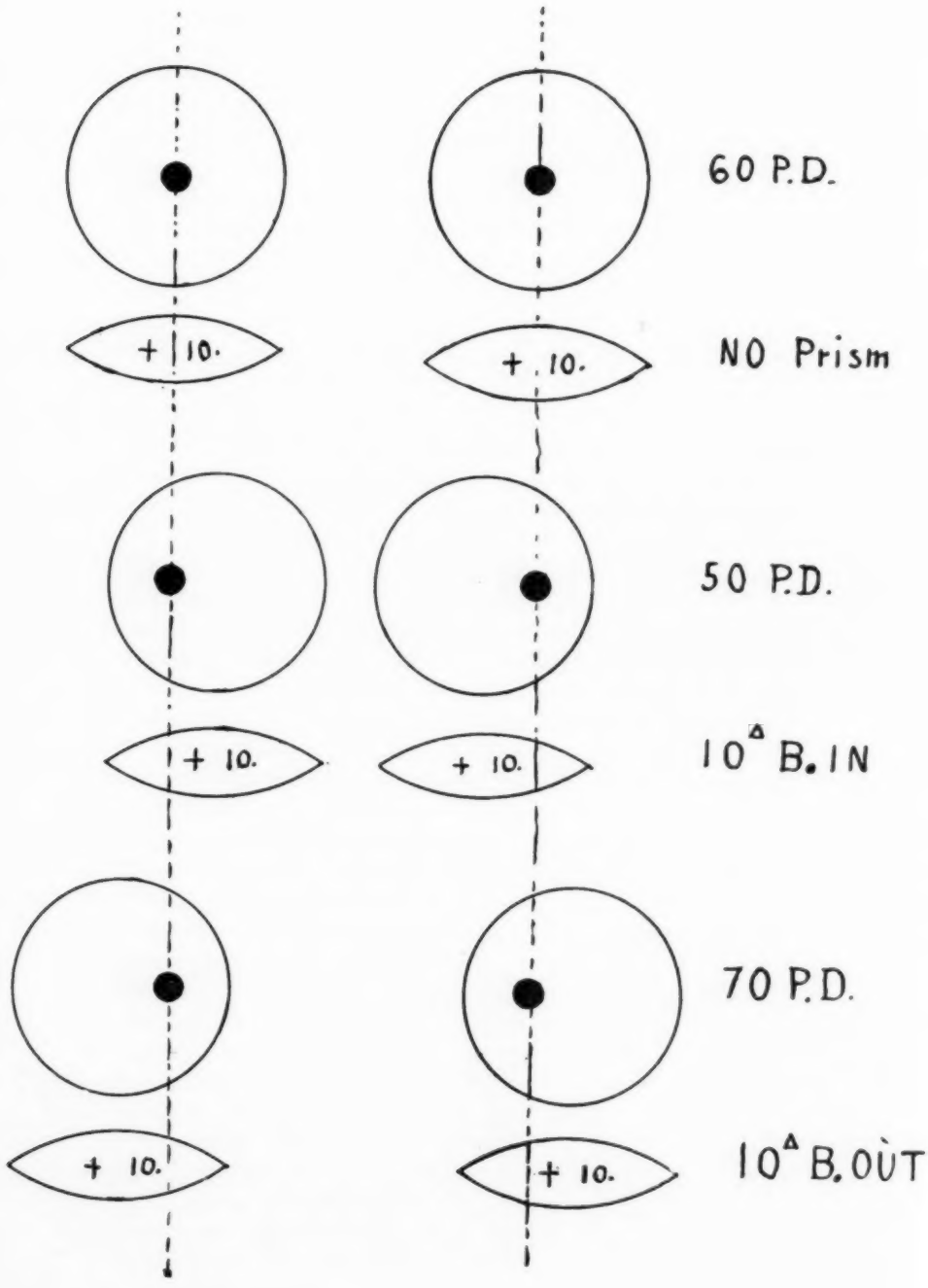
The Decentering of Spheres: The writer is indebted to Javal for the idea of insinuating the prism by decentering the spheres. With + 10 lenses at 10 cm., each mm. of decentration is equivalent to 1$^{\Delta}$, and with the regular size trial lenses, 10$^{\Delta}$ to 15$^{\Delta}$ can be developed by increasing the pupillary distance of the phorooptometer (Fig. 1).

The patient will hold the image fused by this method of introducing prism base out so very much better than by turning the revolving prisms or approximating the half pictures, that it is the *keynote of success.*

The fixed amount of prism is increased by turning the revolving prisms while the patient's eyes are closed, and the + 10 lenses decentered *in* as much as possible. Then the decentering *out* of the spheres is repeated. When the patient can overcome the 60$^{\Delta}$ of the revolving prisms, extra prisms from the trial case are inserted in the clips. In this way 90$^{\Delta}$ can be exhibited.

For home work, as the convergence increases, prisms of 5$^{\Delta}$ and 10$^{\Delta}$ are inserted in the clips of the stereoscope which the patient uses. The average patient becomes keenly interested and *makes the effort* both at his office lesson and at home. He *can note his progress* himself.

At each treatment, the patient's prism duction is tested with loose prisms held before the eyes, but he is never given prisms to use at home. This practice was discontinued many years ago, as it was discovered that some allowed an eye to diverge, suppressed the image, and thus developed a bad habit. The patient's eyes are watched while he is fusing the distant light, in order to detect divergence without diplopia. Fifteen


60 P.D.
+ 10.
+ 10.
NO Prism
50 P.D.
+ 10.
+ 10.
10^Δ B. IN
70 P.D.
+ 10.
+ 10.
10^Δ B. OUT

minutes is enough time for giving a lesson, and the home work required is limited to fifteen minutes, two or three times a day. At the beginning, the patient is seen three times a week, then twice a week, then once a week, the interval being lengthened as progress warrants, so that the twelve or fifteen treatments required will consume about two months.

Marked Photos or Stereographs: To overcome the monotony and to keep up his interest, the patient procures a dozen or more good stereographs, and these are marked with two dots on one side and one on the other, so placed that when

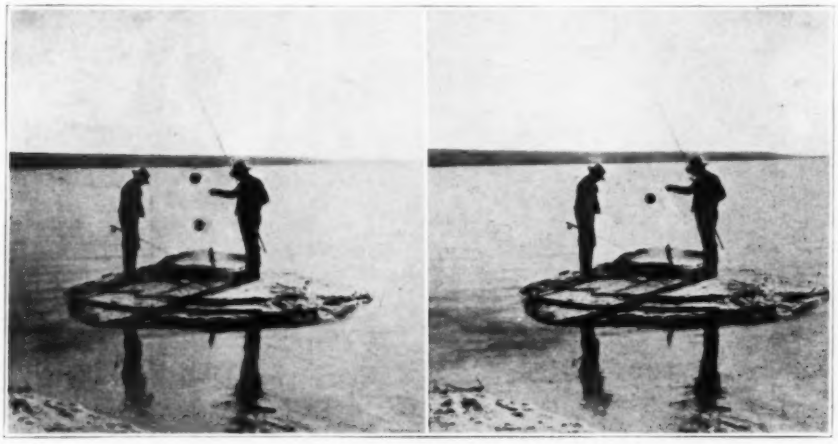


Fig. 2.

properly fused, the three dots appear in a vertical line. This is to guard against a possible suppression of either eye (Fig. 2). A stereograph so marked is illustrated by Landolt in Norris and Oliver's System, Vol. iv., p. 107. Such pictures for home use, with a pair of 10^{Δ} prisms, bases out, in the clips, cultivate fusion and convergence.

Bar Reading: While it is easy to demonstrate defect of fusion with the stereoscope, it is impossible to know to what extent the patient is suppressing one eye in his ordinary reading. To guard against this, Javal constructed a grill of a number of bars, which rested on the reading matter on four legs. As this was a somewhat clumsy contrivance, the writer made a control device, consisting of a strip of aluminum half an inch wide, to be held about five or six inches in front of the face by means of an ordinary head band like those used for holding a mirror. This the patient uses for all reading, and with it in place he must, at least once in every line, use both eyes (Fig. 3).

In many cases a triple control (Fig. 4) is used, but ordinarily the single strip suffices. The use of this is required for some months after the office visits have ceased. Though annoyed by it at first, many patients come to recognize a distinct help in steadying the reading.

Results.

In advocating a special method of treatment, it is realized that something must be said about results, but it is a somewhat difficult task to report accurately concerning therapeutic accomplishments.

Fig. 3.

Fig. 4.

In 1917 the writer[8] published a schedule (Fig. 5) which it was hoped would be generally adopted, so that it might be possible to judge the relative value of different methods of treating heterophoria.

At this time, 236 consecutive cases, which had been treated some years previously, were plotted on this schedule with the following result: (75% cured or relieved).

155 cases of exophoria at distance:

70	"	"	"	or 41% symptoms cured.
82	"	"	"	or 34% " relieved but not cured
14	"	"	"	or 8% " not relieved.
19	"	"	"	result not stated or treatment stopped

Case No	SYMPTOMS H=Headache P=Pain in Eyes B=Blur C=Confusion	EXOPHORIA				DUCTION				Fusion Faculty	No. Treats.	SYMPTOMS C=Cured >=relieved not >	PHORIA				DUCTION				% Loss after 8 mos.	REMARKS
		Phorom.		Cover.		Revolving Pr		Loose Pr.	Stereo.				Phorom.		Cover.		Revolving Pr.		Loose Pr.	Stereo.		
		D	N	D	N	Ad.	Ab.	Ad.	"ONE"				D	N	D	N	Ad.	Ab.	Ad.	"ONE"		

Schedule for Reporting Heterophoria Cases. Proposed by DAVID W. WELLS, M. D., F. A. C. S., Boston.

Fig. 5.

—record incomplete. Assuming that these were not relieved, it would make the number of failures 33, or 21%.

81 cases of insufficiency convergence treated:

36	"	"	"	"	or 44% symptoms cured
34	"	"	"	"	or 42% " relieved
7	"	"	"	"	or 8% " not "
4	"	"	"	"	result not stated or

treatment stopped—record incomplete. Assuming that these were not relieved, it would make 11 failures or 15%.

There have been some relapses (just how many it is impossible to say), but the great majority of the "cured" cases have retained sufficient power for all practical purposes. Patients are admonished to test themselves from time to time with the stereoscope and cards, and if they notice any loss of power to renew the home exercises.

During the last twenty years, the writer has had the pleasure of demonstrating this stereoscopic method to many ophthalmologists from various parts of the country, but very few have ever given it a serious trial. Many have been appalled by the amount of time required. Others have suggested they would like to delegate it to their assistants. Some have asked that their office nurses might be instructed in the technic. This attitude seems to minimize the importance of heterophoria as a cause of eyestrain. What is the trouble with the "rounder" who goes from one man to another, to be given 0.25 difference in the strength of his lenses, or 5° change of axis? It is admitted that the nervous instability, (whatever that may mean) accounts for part of them, but unless the writer is blinded by his enthusiasm, heterophoria and defective fusion, or convergence insufficiency is the cause with the majority. If this be true (and their number is legion) are they not worth curing?

Dr. Jackson says it is essential to "get the patient to make the effort", and who is best qualified to do this? Certainly not the assistant or the office nurse. The man whom the patient has been led to consult is the one who can do the most for him. The patient should receive his individual and undivided attention. There should be no third person in the room, and no distracting interruption. With the phorooptometer stereoscope one is able to watch the patient's eyes while the lenses are being decentered, and if either eye is seen to fail to fix, the patient should be called up sharply

with the words: "Careful, keep that left eye on the dot," etc. He will often express surprise that his lapse has been detected, and renew his effort to fix his attention. Besides he should be encouraged to try a little harder next time, and commended when it is evident that the effort is being made, and he should have no doubt that *his success is a serious matter with the physician.* In short he should be given ahypnotic suggestion for all it is worth.

BIBLIOGRAPHY.

1. Wells. Archives of Ophthal., Nov. 1, 1920.
2. Gould. Ophthalmology, Oct., 1904.
3. Landolt. Amer. Journ. Ophthal., Feb., 1923, p. 96.
4. Tscherning. Physiologic Optics, p. 336.
5. Hazen. New Findings in Ophthalmology, p. 23; also, Arch. of Ophthal., Jan., 1920.
6. Lancaster. Trans. Section on Ophthal., A. M. A., 1923.
7. Jackson. Amer. Journal Ophthal., April, 1922.
8. Wells. Journal of Ophth., Otol. and Laryng., Dec., 1917.

DISCUSSION.

DR. EDWARD JACKSON, Denver: We are fortunate in having such a paper brought before us, in that, while we have generally had a conception that this matter of the development of the fusion faculty was important, it has scarcely been brought into the realm of our practical daily work. This paper does bring it within the realm of practical work, in suggesting means that are necessary and yet practical.

What is the "fusion faculty?" What can we compare it with to understand it better? I think one of the best comparisons that can be made would be with the faculty of standing on our feet, the foundations of which are congenital, inherited, and which faculty of standing erect still has to be developed in every individual. It consists of both sensory and motor elements, sight and the sense of standing; and the sense of fusion depends, ultimately, on the relations established between sensory and motor activities. The original work done, particularly by Dr. Howard during the late war, shows to what extent the sight element has to be cultivated, how perfect it can be in some individuals; and how there are all grades of approach to perfection, as I suppose there are all grades of ease and accuracy in standing. Perhaps an individual can experiment on himself and find the different grades of ability to stand, by properly conducted physiologic experiments. I think, if we get away from the ordinary nomenclature of the books in our discussions and think of "fusion faculty" a little differently, we will be able to appreciate this paper and get some new light on it.

I believe—although this might be a subject of further investigation—that the fusion faculty on the sensory side is concerned strictly with the impressions that are made on the macula and on the fovea. Binocular diplopia is the perception of objects by the fovea of one eye and some other part of the other. We are constantly having a diplopia outside the fixation point, and that diplopia gives us our sense of depth in the general visual conception of our surroundings. It does not bother us. It serves

us if it is not too extreme, and if it does not come into the small region of foveal perception. Remember that foveal vision, as to the human race, is a development that is comparatively recent. Most of the mammals do not have anything of the kind; many of them have practically no binocular vision of any sort. It is only monkeys and human beings that have anything like a developed foveal vision, or anything like a developed fusion faculty. What the fusion faculty of birds is, with their double macula, we can scarcely conceive. It is perhaps as good or better than our own, but totally different. Foveal vision, being a recent development in the history of the race, is particularly likely to be imperfect, and it is certain to vary greatly among different individuals in the completeness of its development and in its accuracy.

So much for what the fusion faculty is. What about our improvement of it? I think that the methods of training the fusion faculty, improving our binocular vision, our power of attaining and maintaining binocular vision, depend to a very considerable extent on analysis of the different parts of this process, that are grouped under this name of fusion faculty. When we change some one element and in that way bring it under the influence of voluntary effort, we change the amount of convergence, and so exert greater effort in the direction of convergence, or somewhat inhibit the convergence while we are taking the exercise. To my mind that is the meaning of exercise with the stereoscope and also with prisms.

I think a method that has not been appreciated in this direction is the one suggested by Priestley Smith, as a means of diagnosis rather than a means of fusion training, the fusion tubes as he called them. They consist simply of two tubes, with one eye looking through each tube, and at the other end of the tube is a slit which makes a line of light, and a dot; and the point is to bring the two slits together so there appears only one; and yet you know that you have binocular vision because there are two dots, one on each side of the slit. These dots can also be brought together and have one dot with a slit on each side.

These tubes give a very simple set of impressions, and yet one that will guide us to the exercise of the motor element of fusion in all directions with great accuracy. Suppose the slit is placed vertically; we make a lateral adjustment and bring the two lines of light into one. The vertical movement can be adjusted by noticing whether the dots are on the same level, or by fusing the dots and leaving the slit double. Then by twisting these tubes before the eye, we bring in the matter of rotation. I think it is about the only possible method of simple and exact exercise of the power of wheel rotation of the eye. All of these things can be done with this extremely simple apparatus. With it, you can get very young children to do exactly what you want them to do, and make sure they are doing it. But it is also a means that is valuable for adults.

Two or three things that have been emphasized in Dr. Wells' paper I want to reemphasize. One is the importance of training the patient to do these things, by bringing to his assistance all the moral support, all the influence that can be exerted on him for the time. I can conceive of this being not only time consuming work, but work which requires

an intense effort on the part of the ophthalmologist, who is trying to train the patient. He must do what we find the hardest thing to do often—to master the will of the patient and make him desire to do the thing he is there to do, and then reinforce his will to do it with all the strength and concentration of which he is capable.

I want to add my plea to that of Dr. Wells for the personal attention of the surgeon. Javal is credited with saying that every case of strabismus could be cured by orthoptic training; and also that it is very likely it was not worth the effort. If a case is to be cured, it would only be cured by effort on the part of the patient, and on the part of the medical advisor, and perhaps the effort of the whole family. This may not be worth the effort, but if you are going to be successful along that line, you must give the effort to those cases in which success is worth attaining.

Dr. Alexander Duane, New York: In considering the question of fusion training, I think it is of importance to discriminate in the first place between cases in which such treatment is evidently applicable, and those in which it is not. In the latter class, we should comprise cases of deviation due either directly or indirectly to congenital anomalies. In such cases, it is usually a waste of time to begin with orthoptic treatment. The latter is effectual only after the basic condition, that is, that part of the deviation due to the congenital paralysis, has been ameliorated by operation.

In one slight matter, I feel that I must differ with Dr. Wells. I confess that it has not been my experience, that when a perfect or even approximate cosmetic result has been secured by operation, the patient has much annoyance from the residual uncorrected trouble.

I quite agree with Dr. Wells, that in heterophoria and squint, whether operated on or not—and particularly perhaps in the operative cases—we should by all means aim to develop the fusion or cordinating faculty, and further, that what we get out of prism exercises is just this and not any strengthening of the muscular power per se.

I must admit, with some reluctance, that my experience with stereoscopic training and with the amblyscope, as well as with other forms of fusion exercises in squint, has been at least somewhat disappointing, and while I constantly use these measures—stereoscope, amblyscope, bar reading, contrasting colors, converging and diverging prisms—and would urge their application in every suitable case, I feel that a squint which does not yield to refractive treatment thoroughly and persistently applied, supplemented by suitable training of the amblyopic eye, is rather likely to come to operation. After operation, when the eyes are approximately straight, I believe these methods come in especially for application, and that probably more can then be expected by their use.

The above remarks do not apply to heterophoria, in which, especially in convergent anomalies, I find orthoptic training of considerable value.

In conclusion, I wish to say that we owe an immense deal to Dr. Wells for his development of the subject, and for the very practical means that he has evolved of stereoscopic training and of bar reading.

Dr. Luther C. Peter, Philadelphia: In making his plea for fusion training, Dr. Wells has opened up many interesting points for discussion.

Routine muscle studies made in every case of refraction confirm his statement, that it is often difficult to determine whether ametropia or heterophoria is the dominating cause of asthenopia. It follows, therefore, as a natural corollary when heterophoria is present, careful refraction and correction of the ametropia should be followed by correction of the heterophoria. It is not my practice to employ the stereoscope routinely in the correction of all forms of heterophoria, but I do find it of the greatest help in esophoria, in a limited number of exophorias, and in certain types of more or less total incoordination of the ocular muscles due to an unstable nervous system.

In hyperphoria, little can be accomplished by any form of muscle gymnastics, and one will save time and aid materially in the recovery from lateral imbalance, by the incorporation at once of prisms in the vertical to correct the full amount of the hyperphoria for distance, as this usually is less than that found at the near point.

In most exophorias, my own experience with prism exercises in the office, continued daily by the patient at home, has been happier than that of Dr. Wells. Exophorias of from eight to ten degrees at six meters, and corresponding exophorias of from twelve to eighteen degrees at the near point, can usually be lessened and the patient made comfortable by proper prism exercises. A limited number of patients, usually those whose abducting power is rather high, sometimes, in my experience, require a combination of prism exercises and the use of the stereoscope.

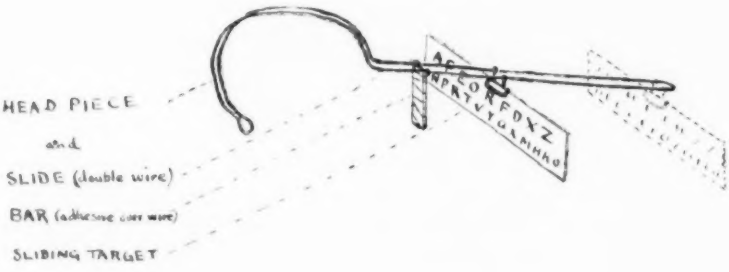
The most difficult cases, however, which we are called upon to treat are the esophorias. In this group of cases, the use of the stereoscope offers the best results. Dr. Wells' method is not as complicated as it seems. If, however, one has not learned his technic, excellent results can be obtained by beginning the treatment with the Wells' stereoscopic cards, and after the patient has thoroughly grasped the underlying principles, or to vary the monotony, ordinary stereoscopic pictures may be substituted.

In the use of prisms or stereoscope for exophoria, or the use of the stereoscope alone in esophoria, the keynote of success is thorough attention to detail and the hearty and energetic cooperation of the patient. As Dr. Jackson has pointed out, "The patient must make the effort."

In the closing statement of Dr. Wells, I most heartily agree. Fusion training is too important and too technical to be relegated to an assistant or to a nurse. Results can be obtained only by one who has enthusiasm and a thorough knowledge of the various steps required by each individual case. The end results and the gratitude of patients who have obtained relief are worth the effort required by the physician himself.

Dr. Sylvester J. Beach, Portland, Maine: With the cards and Dr. Wells' effective method of using high spheres, I sometimes succeed in developing fusion at close range in patients who are still unable to get binocular single vision at distance. This can occasionally be overcome by use of an apparatus on the principle of Dr. Wells' bar. Apparently, this difficulty is psychic rather than mechanical, and can be handled by withdrawing the charts slowly to a distance while the bar is in place. As the patients are children, to prevent peeking an

attachment is required, which will hold the charts in line with the bar. I should think Dr. Wells' headband would be more comfortable than mine.



Dr. Robert Scott Lamb, Washington, D. C.: There are always two sides to a question, and I think we ought to look at both. I do not want to be misunderstood. I am heartily in accord with Dr. Wells. I have even taken the trouble to try to produce, and have produced, fusion in albino nystagmic strabismic youngsters, and if there is anything harder I do not know what it is.

I want to call attention to what seems to be a rather general practice among men, from all I can hear from physicians and patients—the prescribing, after careful examination, of certain lenses, and then leaving it up to the optician to get the lenses in perfect position before the face, and be sure that that lens will do the work. I think, after we go to all the trouble of trying to find out exactly what is needed by very careful measurements, we might just as well give a little more trouble, and see the lenses on the face of the patient, after they have been made up and turned over to the patient by the optician. I know certain men who check up the lenses, that is, they have the patient bring the lenses back and check them to see if the curve is correct, and then they hand them to the patient and say, "Go ahead, this is all right." But they do not see the lens on the face, and I believe with Dr. Duane, that we ought to check up all our work carefully, and I believe by doing so we will find fewer cases in which there is necessity for prisms or necessity for fusion training.

In regard to esophoria, it is associated with a vagotonia in many instances, so the mental processes of the individual are to be considered. She is enthusiastic about something and continues to be enthusiastic about that thing, and may be unable to overcome the esophoria no matter how much effort is made to create a fusion sense, so long as vagotonia remains.

CONGENITAL ABDUCTION DEFICIENCY.

HAROLD GIFFORD, M.D.

OMAHA, NEB.

While this condition has been described so often that it receives a brief notice in most text books, ideas as to its etiology are still widely at variance. There is also marked divergence in the nomenclature of continental as opposed to English speaking writers. In the continental literature, which is chiefly German, the cases are generally referred to as instances of congenital retraction movements, congenital enophthalmos, or some similar title. Kunn,[1] who made the first comprehensive study of congenital anomalies of motility, used the title Congenital Deficiency of Motility. The title which I have adopted is a shortening of the more completely descriptive title used in Duane's[2] classical paper, and will be referred to for short as C. A. D. I find a justification for basing the shortened title on the abduction deficiency in the fact that the latter is the most persistent and most important single feature in these cases, as well as the one to which the next most important symptoms (retraction and deficient adduction) are probably, in many cases, secondary.

MONOLATERAL CASES.

Although the monolateral and bilateral cases of C. A. D. merge to some extent, as will be later indicated, so many cases of the latter have features which are practically absent from the former, that the monolateral cases, which are much more numerous, will be considered separately as the type. The affected eye usually turns in slightly; but in quite a number is parallel with the other or even, in a few cases, turns slightly outward when the unaffected eye looks straight ahead. There is generally slight static enophthalmos. On attempted abduction, the eye generally goes to the median line or a little beyond it, but no farther; and the fissure usually widens slighly and the eyeball comes slightly forward. On looking either up and out or down and out, the eye generally turns a little farther out than when the attempt is made to

turn it straight outward. Adduction is generally somewhat, and often very much decreased; and in the great majority of cases is accompanied by a distinct narrowing of the palpebral fissure with retraction of the eyeball. In perhaps the majority of cases the eye turns up more or less on extreme adduction, in a few cases going straight up instead of inward. Upward and downward movements are generally given as normal, though slight reduction is not uncommon. In quite a number of cases the other eye shows slight deficiency of abduction or adduction, or even, if the range of motility of the other eye is normal, extreme lateral movements produce slight nystagmus.

A large majority of the reported cases (more than 80%) are left sided, and nearly twice as many females as males have been reported.

Another feature, not hitherto noticed, so far as I can learn, is that among the males reported, the predominence of lefts over rights is not nearly so great as among the females.

The vision of the affected eye, in the majority of cases is as good, or nearly so, as that of the other; and unless there is a marked visual defect, stereoscopic vision prevails in that part of the field within which the defective laterals permit free motion. Diplopia is rarely complained of; in nearly all cases, however, where the vision is good, it can be demonstrated with the red glass and prism in the outer field of the affected eye. The absence of marked secondary contracture of the internus is also noteworthy. These visual peculiarities alone would mark off unilateral C. A. D. from the ordinary cases of convergent strabismus.

Anatomic Conditions. The anatomic conditions underlying this syndrome have been exposed to some extent by a number of operations on both the external and the internal rectus. With very few exceptions, the external rectus has been found to be represented by a fibrous band containing no muscular tissue; the internal rectus has sometimes been found to be attached somewhat farther back than normal, or to have one or more secondary attachments farther back than the primary one. In describing these operations on the internus, the operators usually make no comment on the condition of the muscle itself, the inference being that it has been found to be not especially abnormal; but in at least one case, a bilateral one (Birch-Hirschfeld), the internus

was found to be a fibrous, nonmuscular band quite like that found in the place of the externus.*

Exceptional variations in the condition of the external rectus have been found. Bahr[3] could find no trace of an external rectus. Axenfeld and Schürenberg[4] found in place of the external rectus a somewhat fibrous, but not entirely inelastic band. Green[5] found the externus attached to the sclerotic from the usual point of insertion back to the neighborhood of the equator, from which point two fibrous bands led back into the orbit.†

Frequency. In 1905, Duane could find, including 6 seen by himself (and 5 bilaterals), only 54 cases of C. A. D. Birch-Hirschfeld,[6] in the second edition of the Graefe-Saemisch Handbuch, under the title Congenital Enophthalmus and Retractio bulbi, published in 1907, reported only 50 cases. From the literature, I have gleaned enough others to bring the cases hitherto reported up to 105 monolaterals (and 35 bilaterals). To these I can add 30 monolateral cases (and 9 bilateral ones) all from my own practice except one, which has been reported to me by Dr. Brown Pusey. This does not mean that either the size of my practice or my clinical discernments has been anything unusual; but that since the early 90's, when I saw my first case, I have been on the lookout for others and, furthermore, such cases are not so uncommon as one would think from the reports. In discussing the subject with men in different parts of the country, I find that a large proportion of them, on hearing the characteristics of the affection, realize that they have seen one or more cases.

Distribution of C. A. D. The most striking feature in the statistics of monolateral C. A. D., is the prepond-

---

*By testing the extent to which the eye could be rotated inward and outward by fixation forceps, it has been found in several cases, that it could be rotated inward little or not at all—not so much, even, as outward. This indicates such a decided pathology of the internus, that, in the future, as many cases as possible should be tested in this way.

†The only postmortem examination reported on any case of anything like C. A. D., is in the case of Heuck (Kl. Mb. f. Aug. 1879, p. 256) concerning which I agree entirely with the opinion of Duane, that it should not really be classed under the head of C. A. D., as the subject in this case had an affection of a distinctly family type quite different in its main characteristics from C. A. D. These cases are bilateral with marked ptosis and absolute resistance to any effort, active or passive, to move the eyes upward. Axenfeld-Schürenberg have reported a case of this kind. I have seen one family of the sort and among others one has been reported by Lawford (Tr. Oph. Soc. Un. Kgd., VII, 262).

erance of left eyes affected. Of the 135 cases collected by me, including my own 29, 101 were left sided, and a somewhat similar proportion appears in all the different series of any size, which have been reported. Out of 26 by Evans[7], 84.3% were lefts. In 14 cases added by Duane, 78.5% were lefts. In my own series (including one by Pusey) of 30, 83.4% were lefts. Hardly less striking is the preponderance of females over males. In the 70 cases comprised in the lists of Evans, Duane and myself, 65.7% were females. In the entire list of 135 which I have collected, 57% were females. As to the larger proportion of rights among males than among females, of the 46 women in the above list of 70, the proportion of lefts was 91.3%; while of the 24 males, 66.7% were lefts. Of the whole series of 135 cases, females showed 83.3% of lefts, while the males showed 63.1% of lefts.

Explanation of Preponderance of Females. The prevalence of females over males, which has been observed in every large list of cases of C. A. D., has always seemed to me to be most probably the result of the greater attention which is generally paid to the personal appearance of girls and women. In a large proportion of cases of C. A. D., the deformity is so light, when the patient looks straight ahead, as to attract no attention, and in greater degrees it is apt to be concealed to some extent by a slight rotation of the head. The consequence is that the majority of these patients do not report for treatment until they are well grown and, frequently, they attract so little attention even then, that they are only accidentally discovered by the oculist. The belief in the personal appearance explanation of preponderance of females in C. A. D., has already been expressed by Hoefnagels.

Explanation of Smaller Proportions of Lefts Among Males. This peculiarity is evident in all the different lists that I have studied, but as far as I have observed, it has not received special comment. In my combined lists, the females show 83.3% of lefts, while the males show 61.1% of lefts. As I know of no obstetric or anatomic explanation for this differences between the sexes, it seems to me that an occupational explanation is the most reasonable one. There is probably just as large a proportion of left C. A. D. among males as there is among females, but among boys the right sided cases are more apt to be noticed and brought up for treat-

ment, because the vast majority of them, being right eyed, games and occupations which occupy their time are much more apt to call attention to right eyed defects than to those of the left eye. With the increasing use of the mirror by the boys, this sex difference should tend to disappear from the statistics of C. A. D.

Explanation of the Symptoms. The lack of abduction is easily explained by the anatomic conditions found. The enophthalmus with retraction of the globe and narrowing of the fissure on adduction has been the subject of the greatest controversy. By perhaps the majority, the rigidity of the external rectus has been held to be sufficient explanation; while others fall back upon an attachment of the internus too far back or upon the presence (demonstrated in several cases) of secondary posterior attachments of the tendon; or upon the assumed atavistic development of a retractor bulbi with an attachment near the posterior pole. Peschel,[8] Parker,[9] and others attribute the retraction to the action of the superior and inferior recti in their attempt to supplement the deficiency of adduction. Treacher Collins[10] suggests that perhaps, in addition to unusual attachments of the internus, defective check ligaments permit an unusual retraction effect from the recti. A similar theory has been advocated by Varese.[11] The slight (relative or positive) exophthalmus on attempted abduction to which Wolff,[12] in particular, called attention is probably due to the combined action of the obliques. The tendency for the eye to turn up and in, or more rarely down and in, on attempted adduction has been explained by Wolff as an effect of the resistance to retraction offered by the optic nerve, while Duane regards it as evidence of oblique spasm. The pros and cons of these different theories have been so thoroughly thrashed out in the series reports of Axenfeld and Schürenberg, Evans, Duane and Birch-Hirschfeld, to say nothing of numerous other reporters of one or two cases, that I shall not attempt to go into them deeply, since my main object in writing this paper is to discuss the etiology of the condition with especial reference to the birth injury theory. With this object in view, I shall give but scant attention to the individual peculiarities of the cases of C. A. D. which I have collected from the literature, in addition to those included in the lists above noted, or to those of the 29 unilateral cases from my own practice.

Etiology. The chief interest at present centers in the question of etiology. Kunn, who wrote most extensively on congenital defects of the eye muscles, considers them the result of aplasia, but whether primarily of the muscle or of the nucleus, he does not like to say. Axenfeld and Schürenberg, Evans, Duane and Birch-Hirschfeld attribute the conditions to peripheral defects of the muscles and their insertions, without anything fundamental as to the reasons why such peripheral defects should occur. Moebius, for no apparently valid reason, believes they may be examples of intrauterine nuclear degeneration (Kernschwund). Peters believes the cause to be a defective anlage of the muscle.

Birth Injuries as a Cause of C. A. D. In 1893, Parent[14] expressed the belief that many congenital eye muscle defects were due to birth injuries; though whether he had in mind orbital or brain injuries can not be told from the context. Birch-Hirschfeld, though in general favoring muscular abnormalities as a cause of C. A. D., admitted the possibility of orbital hemorrhage from birth injury as a cause of "congenital enophthalmus."

In the late 90's, the writer, impressed by the preponderance of lefts over rights, reached, and taught to his classes, the conclusion that unilateral C. A. D. was probably a birth injury from pressure, but did not give the matter sufficient thought to work out the modus operandi of the process, as was done by Gallus[15] and Hoefnagels[16]. The former, in 1910, merely expressed the belief that C. A. D. was due to a birth injury with scar fixation of the externus; while in 1914, Hoefnagels called attention to the remarkable agreement between the proportion of left and right C. A. D., when compared with the relative frequency of the left occipito-anterior position as compared with other presentations. He pointed out, that from the L. O. A. position, the left temple, in the rotation of the skull, would be forced against the promontory; the results, perchance, being an anemia which, if long continued, might cause the degeneration of the externus muscle into the fibrous band which is commonly found to take the place of the muscle in these cases. He cites Leser's experiments to show that a pressure anemia of two to three hours produced a myositis followed by nearly complete regeneration of the muscle cells; while if the pressure was continued for five to six hours, the greater part of the muscle underwent permanent fibrous degeneration. He

also quotes de Vries, who found in normal births, without external signs of injury, 11 per cent with retinal hemorrhages.

Stirred up by Hoefnagels' paper, and later by attacks on his theory by Eichmann[17] and Peters,[18] Gallus elaborated his original suggestion, explaining his belief that C. A. D. is due to rupture of the muscle sheath of the externus by intrapartum pressure, with subsequent degeneration of the fibers. Both he and Hoefnagels emphasize their belief that instrumental injuries are not to be looked for as the main cause of C. A. D., but rather relatively normal deliveries in which, for one reason or another, the outer side of the orbit or eye has been subjected to unusual pressure.

Turning now to the actual evidence of birth injuries in cases of C. A. D., we find very little that is positive. Bloch[19] has reported three cases of fairly well marked scars on different portions of the face and head. One of my cases had the same sort of history, with a well marked forceps scar in the parietal region on the corresponding side. It is by no means certain, however, that in these cases, or in the small number of others in the list in which forceps were used, damage to the eye was the result of the use of the forceps rather than of the general severity of the pressure to which the head had been subjected before the forceps were used. In the great majority of reports of C. A. D., either nothing is said of the character of the birth, or the latter has been described as normal.

It is evident that if birth injury is the main cause of C. A. D., it must generally be produced by the natural forces in such a way as to give very little external evidence of the injury. The comment of Dr. Davis (see below) is especially enlightening on this point. It should also be remembered, that in the case of Hofmann, where one eye was squeezed out of the orbit, the depression of the frontal bone entirely disappeared in the course of an hour.

Obstetric Inquiries. Inquiries among prominent obstetricians gave mostly negative results. They were perfectly willing to concede the probability of injuries to the eye muscles from birth pressure or hemorrhages, but never having had their attention called to C. A. D., they had paid no attention to the comparative frequency of slight injuries to right or left temples in ordinary births or in the different presentations. My attempt to subsidize investigation on the part of

internes and nurses on this point received no encouragement from the hospital authorities of one of our largest maternities, and produced practically no results. Several gentlemen were kind enough to answer my inquiries at some length. Dr. Wilber Ward, of the Sloane Maternity in New York, wrote that "in the L. O. A. position, there is a larger number of slight marks due to forceps at the outer corner of the *right* eye. On the other hand, the posterior occipital positions (mainly R. O. P.) are by far the most difficult instrumental deliveries, and the marks from them are more likely to be on the *left* side"; further, that injury about the temple region aside from forceps marks is practically unheard of. Further he says, "I can see no obstetric reason for the unusual percentage (in C. A. D.) affecting the left eye, and I do not believe that the sacral promontory exerting pressure in the L. O. A. position would explain it. There certainly can be no relation between this pressure and the large proportion occurring in females. The latter fact, if substantiated in a large series of cases, would, of itself, it seems to me, rule out birth injuries [valid if there were no other explanation for the preponderance of females. G.]. I also doubt very much whether there is any particular difference in the number of slight contusions, etc., occuring during birth on the right and left sides respectively. I have no impression of such a state of affairs in my obstetric work. Impressions, however, are misleading . . . . ." Dr. Joseph B. De Lee, Chicago, writes "The explanation of paralysis of the muscles of the eye does not appeal very strongly to me. I would be more inclined to think that it is due to hemorrhage in the nerves or in the ganglia. I have for many years entertained the notion that most of the minor paralysis of the eye muscles and of the facial muscles are due to central or peripheral nerve injuries inflicted during birth. I do not think that the left side of the head sustains greater damage during labor than the right side." On the other hand, Dr. Edward P. Davis, Philadelphia, writes, "Pressure injuries to the fetal cranium and face are much more common than is usually supposed. Unless bruising or laceration occurs, such conditions are commonly not detected. I have seen numerous instances where, after a particularly long and difficult labor, accompanied with birth pressure, some deviation in the eyes subsequently developed. As regards the frequency of this condition in the left eye, in the majority of

labors the left side of the face is directed toward the promontory of the sacrum. No pelvis is absolutely normal in contour, and there is no way of knowing in a given case that pressure may not develop in this region. The fact that in the history of the case it is stated that nothing unusual occurred at the time of birth, is of no value whatever. The majority of the obstetric cases are attended in such a loose and careless manner that their histories are worthless."

Such slight testimony as we have from German obstetricians is more favorable to the birth injury theory. Gallus quotes Kramer as his mainstay in bringing out the importance of statistics of the different obstetric positions in support of the birth injury theory. Leitzmann (cited by Bloch) has noticed that the occurrence of edema in the temple region of new borns is more common on the left side than on the right. Along the same line is the testimony of Küstner,[20] who, although he believes that paralysis of the eye muscles from birth injuries are almost always due to hemorrhage at the base of the brain, speaks of a symptom frequently observed in the form of a characteristic pressure furrow made by the promontory on the infant skull, beginning on the parietal, running down toward the ear, and then forward and downward across the temple to the zygomatic region of the face.

If further evidence were necessary as to what the processes of parturition, without instrumental aid, can do to the eye, the famous case of Hofmann[21] should suffice. He was called to a women who had just given birth to a child, and found the infant's right eye hanging out on the cheek, attached only by the inferior rectus and some loose connective tissue. The birth was spontaneous but protracted. Three years later, he was called to the same woman and, on acccount of delayed birth and failing uterine power, delivered the child with forceps without difficulty. As the head was delivered, the right eye of the babe fell into his hands. It was quite certain that the forceps had played no part in the enucleation of the eye, but the frontal bone was much depressed. As the mother died after a third delivery, a section was made which showed the conjugate diameter to be only 3 inches; the junction of the last lumbar vertebra with the sacrum was not rounded off in the normal manner, but presented a sharp angle. It is interesting to note that the first of these children showed a marked depression of the frontal bone, which dis-

appeared within an hour. Nothing was said about the disappearance of the depression in the second child, probably because it died shortly after birth. Several other cases have been reported (De Wecker,[22] Bock,[23] Polliot[24]) in which an eye has been found at birth to be enucleated or nearly so, and it has been assumed that these accidents had been produced by the midwife's finger, but in view of the neatness with which Nature accomplished the enucleation in Hofmann's case, it seems as if it might well be unjust to blame the midwife in these or other cases.

Scrini,[25] examining the eyes of 136 newborns, found 60 with convergent alternating strabismus. This strabismus tended to a disappear, although Scrini could not follow them through. It occurred more frequently in primiparae and in prolonged labor generally, although in many of them labor was not especially prolonged. He gives no figures as to the distribution between males and females.

Radiographic Results. In the effort to obtain information as to obstetric lesions of the orbit without any permanent exterior sign, I had orbits radiographed in 8 cases of C. A. D., with negative results, except that in one bilateral case, the outer wall of the left orbit at the lower part tipped inward slight more on the left side than on the right.

Explanation of Narrowing of Palpebral Fissure on Birth Injury Theory. It is generally accepted that the narrowing of the fissue which accompanies, and is the most obvious sign of the retraction of the globe, is due to the enophthalmus with its lack of support to the lids. Duane, however, believes that this must be due to an action of the orbicularis, because in the narrowing the lower lid comes up to some extent. It seems to me, that while it is impossible to disprove the occurrence of such an associate action of the orbicularis with the internus, the upward motion of the lower lid is so slight that it can be accounted for by the recession of the globe, which in its normal position holds the lower lid downward as well as forward. Moreover, the associate action which we should expect with a defective internus would be widening from stimulation of the levator rather than a narrowing.

In a few cases, the retraction movement so dominates the scene, that it is a question whether the cases should not be classified rather as cases of abnormal external fixation than of abducens deficiency. For instance, in the first case reported

by Wolff, the eye at rest turned slightly outward and could be rotated a little further outward by an effort; while on the attempt to look inward, the eye did not turn in at all, but turned straight up with extreme retraction. The oblique movements upward or downward, which the affected eye frequently exhibits on attempted adduction, have been considered by Wolff to be due to the resistance offered by the optic nerve to directly backward movement.

But if we admit the existence of abnormal insertions or separate posterior attachments of the internus, how shall we account for them on the birth injury theory of C. A. D.? On the other hand, how shall we account for this association of retraction and adduction deficiency with abduction deficiency on any other theory? However much some may be inclined to explain the condition of the externus by an aplasia or by intracranial hemorrhage, these suggestions can hardly be applied to an active but abnormally acting internus, especially when we consider that the internus is the last muscle that we should expect to be associated with the externus in an aplasic deformity. On further reflection, it seems to me that this condition of the internus constitutes the strongest evidence of intraorbital injury from pressure or hemorrhage or both. Assuming that the condition of the external rectus is due to undue pressure, it is evident that the inner side of the globe must also have been subjected to undue pressure, though to a less extent. It does not seem fanciful to assume, that as the result of this pressure, with or without hemorrhage, the internus may have become attached to the globe over an unusually large extent, thus giving the effect of an attachment too far backward; and in some cases such attachments may have been formed separately at points farther back than the main attachment.

Heredity. Heredity seems to play no part in the great majority of cases of C. A. D. In my own list, I have a mother with typical left C. A. D., one of whose children, a son, has bilateral C. A. D., and a similar case is reported by Harlan[26]. Kraus[27] reports a mother with monolateral C. A. D., whose son also was affected in the same way. An instance in which more than one child in the same family was affected is the family of Kunn-Wolff, in which two sisters and a younger brother had left C. A. D. These cases do not necessarily speak against the birth injury theory, for even the cases in

two generations may denote, not a tendency to aplasia of the externus or its nucleus, but the existence of an inherited form of pelvis or of uterine action.

The case of Hofmann, already referred to, in which two successive children were born with the right eye pushed out of the orbit, showed both how birth injuries may occur in more than one member of a childship; and also, inasmuch as the woman had two other children who were not affected in the same way, that there is no necessity for all the members of the same childship being affected in the same way by such a pelvic deformity.

A more serious objection to the birth injury theory is presented by cases in which C. A. D. has apparently been transmitted by the father. Günsberg[28] reports a family in which a mother and son had left C. A. D. Of the son's children, two had normal eyes, while of the other three (all girls), one had alternating divergent strabismus with uneven pupils, another had typical left C. A. D., and the third had bilateral C. A. D.

Waardenburg[29] reports a family with eleven living children. The father and second, fifth and seventh child all had left C. A. D. It must be admitted that such cases create quite a strain on one's belief in the birth injury theory. It has, however, occurred to me that injury to the externus would be favored by an unusually large head. This might be the responsible factor of transmission in these rare cases. Unless I misunderstand Gallus, this is the explanation which he would offer of transmission of C. A. D. through the father. Lest the idea may seem entirely forced, I cite from Bloch; In the family of one of his patients with C. A. D., all four births were unusually difficult, and in one of them at least, this was due to a specially large and unusually shaped head.

Other Explanations of Preponderance of Lefts. The question may be raised in attempting to account for C. A. D., on some other theory than that of birth injury, whether the left externus is not specially subject to intracranial disturbance which would lead to its atrophy or degeneration. That the 6th nerve is especially subject to such influence in other than congenital paralyses seems to be fairly well established. Its relation to the foramen of Dorello, at the apex of the petrous bone, makes it specially liable to be affected by various forms of intracranial pressure, or by meningitis transmitted from the middle ear. Blows on the head also affect

the 6th very commonly. In the statistics of lethargic encephalitis, it seems to be affected more often than any other nerve; but any evidence to show that the left sixth suffers in anything like the preponderance in which the externus muscle suffers in C. A. D. is wanting.

More striking is a personal communcation made to me by Professor Poynter, Dean of the Anatomical Department of the University of Nebraska. In investigating the effect of temperature on chick embryos, he found that where the eggs were incubated for six hours, then put into the icebox for two hours, then incubated as usual, he got about 60% of undeveloped eyes; and of this 60%, 95% were lefts. It is a far cry from a chick to a child, but if anything like such a one sided result could be obtained by prenatal noxae in mammals, the rating of the birth injury theory would certainly decline.

Effects from Intracranial Birth Injury. In the absence of any knowledge of problems connected with C. A. D., most obstetricians who have given the matter of eye muscle paralysis any thought, favor the idea that the latter are due to intracranial hemorrhage. The difficulty with this intracranial hemorrhage theory is that it offers no explanation of the remarkable picking out of the external rectus, or of the combination of such externus injury with injuries or peculiarities of the internus. The same is true, though to a less extent of, such suppositions as that of Will Walter (Arch. of Oph., Mch. 1916) who suggests that C. A. D. may be the result of pressure from bone displacement at the apex of the petrosal bone.

Bilateral Cases.

The bilateral cases of C. A. D. are by no means so well defined a group as the monolateral ones. Some of them show, in each eye, a typical picture of unilateral C. A. D., with absence of abduction power and more or less limitation of adduction, together with retraction movements and free vertical movements. Many of them, however, show a tendency toward restricted mobility in all directions, up to the point of nearly complete immobility. Many of them also show more or less and often complete involvement of both facial nerves. They also show a strong tendency to be complicated with other congenital defects, such as deformed ears, defective or missing hands, fingers or feet, together with defects in the

muscles of the chest. They are not so numerous as in the unilateral cases. I have collected 43 cases, including 9 of my own, having omitted several on the borderline of complete external ophthalmoplegia. The proportions of the sexes represented in this group are in marked contrast to those in the monolateral group. For instance, of the 43 bilateral cases, 25 were males and 18 were females, as opposed to 57 males and 78 females among the monolaterals.

This contrast may be partially explained on the personal appearance theory, according to which, where the deformity is slight, as in most unilateral cases, the girls are more apt to be reported than the boys; but where the deformity is very marked, as it is in all of these bilateral cases, the boys would be reported with relatively greater frequency. This would not, of course, account for the surplus of males, which is perhaps not so large as to require a special theory to explain it, but which, on any birth injury theory, might be attributed to the larger size of the male head.*

It should be noted that among 43 bilaterals, 14 were complicated with facial paralysis. This was bilateral in 10 and monolateral in 4; in 3 of these 4, the left side was affected. The facial paralysis in these cases might, if one were laboring to prove a point, be attributed to an additional birth injury, but in view of the close connection between the nuclei of the 6th and 7th nerve, it must be admitted that this complication speaks strongly for a central lesion of these double defects, especially as in several of the cases the facial paralysis was limited to the superior branch.

It should be noted also, in favor of a fusion of the two groups that on the one hand, several cases generally classed in the monolateral group have had so much disturbance of the motility of the other eye, that some men have classed them as bilaterals; while among the bilaterals there are several cases in which one side was decidedly worse. In hereditary cases, also, the lines between these groups tend to become confused. The majority of these hereditary families, in fact, showed a mixture of monolateral and bilateral cases in the same family. On the other hand, again, the fact, that of the 135 monolateral cases, only 1 or 2% were complicated with

*It would, of course, be expected that the larger sized male head would be more subject to birth injury than the female, but this tendency we assume to be more than outbalanced, in the monolateral cases, by the importance of personal appearance among females.

facial paralysis or other marked bodily deformities, speaks strongly for a division between the groups.

Conclusions.

After a careful consideration of the evidence, the writer is of the opinion that the question of the etiology of C. A. D. still needs a lot of clearing up. The difficulty of getting first hand evidence of the actual conditions at birth, reduces the matter to one of probability. If it were not for the bilateral cases, my conclusion would be, that in spite of the absence of direct evidence of birth injuries in the reported cases, the relative proportion of rights and lefts, together with the combination of defects in two opposing eye muscles, would make it highly probable that the great majority of the cases were due to orbital intrapartum birth injury. Even facing the fact that we have all gradations between the strictly monolateral form and the bilateral cases without complications, the birth injury theory would still appeal to me as the most frequent cause. But when among bilaterals we find cases in which, with the eye conditions entirely typical and uncomplicated, there are also other bodily defects which could by no stretch of the imagination be regarded as birth injuries, we must admit that it is highly probable that something else than a birth injury may cause all the typical symptoms of C. A. D. Perhaps the only permissible attitude is one of not proven on any count. Many of the bilateral cases are almost certainly not the result of birth injury; while the latter may well be the cause of many or perhaps the great majority of the unilateral C. A. Ds.

In planning further study on this subject, it seems to me special attention should be paid to passive (rotation with forceps) mobility of the affected eyes. Second: To "follow through" observations on the eyes of new borns. Third: To experimental work on the effect of pressure on the eyes of young animals, both with regard to the production of degenerations in the muscles as well as with regards to its effect upon the internal structures of the eye, when subjected to pressure sufficient to produce degeneration of the muscles. Fourth: To the study of the effect of injury to the nuclei on the microscopic structure of corresponding eye muscles.*

*Sickness has prevented the writer from giving the abstracts of cases and the completion of the bibliography. These omissions will be repaired in the article as it will appear in the American Journal of Ophthalmology.

REFERENCES.

1. Kunn. Beiträge z. Augenheilk., Heft 19.
2. Duane. Arch. of Oph., 1905, p. 133.
3. Bahr. Jahresbericht f. Oph., 1896, p. 405.
4. Axenfeld and Schürenberg. Kl. Mb. f. Aug., 1901, vol. II, p. 844.
5. Green. Trans. Amer. Acad. Oph. and Oto-Lar., 1913, p. 358.
6. Birch-Hirschfeld. Graefe-Saemisch, 2nd Ed., Krankh. d. Orbita., p. 219.
7. Evans. Oph. Rev., 1903, p. 1.
8. Peschel. Cb. f. pr. Aug., 1904, Jan., 11.
9. Parker. Cited by Evans.
10. Collins. Brit. Med. Journ., 1899, vol. II, p. 846.
11. Varese. Jahresbericht f. Oph., 1901, p. 264.
12. Wolff. Arch. f. Aug., vol. XLIV, p. 80.
13. Moebius. Münch. med. Woch., 1888, pp. 35, 91; 1892, pp. 41, 55.
14. Parent. Cited by Kunn. Beiträge z. Augenheilk., Heft XXI, p. 22.
15. Gallus. Kl. Mb. f. Aug., 1910, vol. I, p. 493; Arch. f. Aug. Lit. Rept. for 1914, p. 293; Arch. f. Aug., vol. LXXXVII, p. 35; Ibid., vol. XCII, p. 31.
16. Hoefnagels. Nederlandsch Tijdschrift v. Geneeskunde, 1914, vol. I, No. 8 (English Trans. by Blaauw).
17. Eichmann. Cited by Gallus, Arch. f. Aug., vol. LXXXVII, p. 35.
18. Peters. Arch. f. Aug., vol. LXXXVIII, p. 192.
19. Bloch. Cb. f. pr. Aug., 1891, p. 134.
20. Küster. Müller's Hdb. d. Geburtshülfe, pp. 288-295.
21. Hofmann. Monatsschr. f. Geburtskunde, 1854, vol. IV, p. 6.
22. De Wecker. Ann. d'Oculist., July, 1896, p. 40.
23. Bock. Cbl. f. pr. Aug., Jan., 1902.
24. Polliot. Arch. f. Aug., Lit. Rep., 1914, p. 133.
25. Scrini. Arch. d'Oph., 1901, p. 248.
26. Harlan. Trans. Am. Oph. Soc., 1885, p. 48.
27. Kraus. Münch. med. Woch., 1905, p. 1957.
28. Günsberg. Kl. Mb. f. Aug., 1889, p. 264.
29. Waardenburg. Abst. in Am. J. of Oph., 1923, p. 44.

DISCUSSION.

DR. ALEXANDER DUANE, New York City: Congenital abducens paralysis is not very uncommon, although not nearly so frequent as congenital paralysis of the superior rectus. I, myself, have seen over twenty cases in which the paralysis was certainly, or almost certainly, congenital. In addition, there have been cases in which there was a weakness not amounting to paresis, of the external rectus, existing probably since birth. Insufficiencies of this latter sort are clinically important, since they may call for operation.

The cases of congenital abducens paralysis fall into several distinct types. First, there is the form, assumed to be due to nondevelopment of the nuclei, in which a bilateral abducens is associated with a bilateral facial paralysis. This is very rare. I have seen no case in my own practice, and but one rather atypical one in the practice of another surgeon. Much more frequent are the peripheral paralyses attributable either to structural malformation of the external rectus or to birth injuries. These peripheral cases comprise:

1. Cases apparently due to structural defects of the muscle itself. These comprise two subdivisions, viz.: (A) Retraction cases, and (B) Nonretraction cases.

(A) The retraction syndrome. In a typical case of this condition, one eye, usually the left, cannot move out at all and moves but imperfectly inward. When it attempts to move inward, it retracts into the orbit and often rolls up, and at the same time the palpebral fissure markedly contracts. The condition is seen most often in females. It is occasionally bilateral. My own observation indicates that the retraction syndrome comprises about 40 per cent of the cases of congenital abducens paralysis. While it has been demonstrated that the cause is not always the same, I believe that in most cases it is due, as Dr. Gifford says, to replacement of the external rectus by an inelastic band, destitute of power to move the eye outward and acting as a restriction to the movement of the eye inward.

(B) Nonretraction cases. Cases of paralysis of the abducens, unaccompanied by restriction of the internal rectus or by retraction of the eye and narrowing of the palpebral fissure in adduction. I have notes of ten cases, four of which were bilateral. In three of these ten cases, there was paresis of one or more of the elevators or depressors; in two there was nystagmus; in one, microphthalmus of the affected eye. In three of the bilateral cases, the paralysis was only moderate; in one it was very marked, and in this there was, consequently, an extreme degree of convergent squint which had been present at birth. In the six unilateral cases, this paralysis was complete in three. In at least two of the others, it appears to be complete, owing to the extreme reluctance of the patient to making an attempt to move the eye outward. In these two there was really a fairly considerable power to move the eye, but only occasionally, and by some special maneuver, could the movement be evoked, nor when the patient did produce it was it maintained. In cases of this structural type that we have operated, no actual defects as paralysis, frail tendons, or adhesions of tendons to capsules have been found.

The last type of abducens paralysis is that noted by Dr. Gifford, namely, the variety due to birth injuries. I have seen two cases which were quite surely, and one that was very likely, due to this cause. One at least of these was marked by the sort of inertia or reluctance of movement just noted, and therefore appeared more complete than it was.

There is no sharp dividing line in aspect between the traumatic and the nontraumatic cases, and I am inclined to think that some I have classed under the latter may be really traumatic. Dr. Gifford's suggestion that the retraction cases are due to traumatism is surprising to me, but is certainly suggestive. In these cases of congenital paralysis, we can usually prove the existence of diplopia, either by the demonstration of double images on the screen, or in the case of the very young subject, by the evidences (head turning, shutting of one eye) that the patient gives of his efforts to avoid diplopia.

The question of treatment is of interest. Advancement of the external rectus is indicated only when there is some evidence of power in the affected muscle. It is definitely contraindicated in the retraction syndrome, where in mild cases, anyhow, it would aggravate the symptoms. In cases where advancement alone promises little, it may be replaced either by tenotomy of the internal rectus, or perhaps by the substitution operation, in which slips from the superior and inferior recti are attached to the insertion of the external rectus and serve as abductors. But this latter operation, again, would hardly be applicable in the retraction syndrome.

Dr. John Green, Jr., St. Louis: I presented this subject to the Academy in 1913. At that time, I was able to report five cases which had come under my observation within the space of three years, and since that time I have seen five or six more. Thus, I am in agreement with Dr. Gifford, that the condition is not as rare as the paucity of published cases would indicate. I believe that it is often overlooked.

I operated on one of these patients, although it was of the type that Dr. Duane says should not be operated, that is, the retraction type, and found the external rectus far from normal. It was a thick fibrous band adherent to the sclera as far back as the equator, and it had two distinct points of insertion. The result of the operation was nil. There was no improvement in the position of the eye.

Dr. J. M. Banister, Omaha, Nebraska: I wish to make one little suggestion with reference to this interesting class of cases which Dr. Gifford has brought up, and it is this: In these cases of birth injuries, what is the injury that does it? If the nucleus has been injured in any way, would we have a condition limited to one side? We all know, that in nuclear injuries, any paralysis is of the conjugate variety and both sides are affected, but in this condition we find only one. So it seems to me, looking at it from a clinical standpoint, that one eye being affected would remove the possibility of nucleus involvement. What then could it be? Those of us who have done obstetrics know that in some cases of severe labor you have a great deal of pressure on the skull; there is a great deal of irritation; you have a delayed labor, and to secure definite labor, you use forceps which compress the head. I would make the suggestion that as the result of such compression in those cases, we have some type of edema resulting. We have an edema

after a meningitis. That edema has been proved by Cushing and others to have a most marked effect upon the sixth nerve. Some have claimed it is owing to its antero-posterior direction and its length, which causes the brain stem to pull on the nerve. Cushing's view, as shown by Dr. Sears' paper, is that the anterior inferior cerebellar artery and the internal artery constrict the sixth nerve, just as if a rubber band were placed around it. Might it not be possible that the edema has such a constricting effect as to disable the nerve and have that condition remain permanently?

In the other case to which Dr. Gifford refers, in which the third nerve is involved, a similar explanation could be advanced. The third nerve comes up between the superior cerebellar and the posterior cerebellar arteries, and when the brain stem is pushed back, the third nerve would be nipped as if it were held by a rubber band.

Dr. Harold Gifford, Omaha, Neb. (closing): I think Dr. Banister's suggestion is very interesting. I thought I had gone over most of the ways by which injury to the external rectus could be produced, but his theory is new to me and may be of considerable value. We must remember, however, that in the condition which we are considering, there is, in nearly all cases, besides the defective abduction, a deficiency or anomaly of the internal rectus, while the others muscles supplied by the third nerve are generally not affected. This picking out of the internal rectus is hard to account for on any theory depending on intracranial pressure as the noxious agent. On the other hand, you can account for it if you assume that these are birth injuries; because when you have pressure on the outer side of the globe, you also have the inner side of the globe pressed against the inner wall of the orbit. This, with the addition of hemorrhage within the orbit, might account for the fixation of the internal as well as the external rectus in so many of these cases.

THE ETIOLOGY OF PHLYCTENULAR KERATOCONJUNCTIVITIS*.

W. W. Blair, M.D.

PITTSBURGH, PA.

Anyone working daily in the outpatient department of a large eye clinic, soon becomes familiar with the picture presented by the poor, ill clad, undernourished mother, holding in her lap an equally sickly looking, unwashed child, whose head is buried in the folds of a filthy shawl, and who whimpers at the slightest exposure to light. From this, one may almost hazard the diagnosis of phlyctenular disease of the cornea or conjunctiva. Upon closer examination, we find the characteristic bleparospasm, swollen lids, and profuse lacrimation: when the lids finally can be separated, we see the grayish area upon the cornea, or the tiny droplet like elevations at the limbus, or the large pinkish-yellow, round elevation in the conjunctiva at some distance from the corneoscleral border. The clinical picture is one that is so familiar to everyone, that there is no need to dwell further upon it.

Proper treatment promptly instituted, is commonly followed by a speedy amelioration of all the symptoms—only to be succeeded by a return of the same in the identical, or the fellow eye after a few weeks or months: in fact, the tendency to recurrence is one of the characteristics of the condition.

There is no doubt that the earlier English and French ophthalmologic writers confused phlyctenular disease with interstitial and other forms of corneal disease, but these confusions no longer exist. We now know the disease as a definite entity, and are concerned today chiefly with its underlying causes.

As evidence of the importance of phlyctenular disease, I would state first, that in our clinic, where we receive an

---

*Synonyms: Keratitis Phlyctenulosa, Keratitis Eczematosa, Scrofulous Conjunctivitis, etc.

average of 2,500 cases each year, the incidence of phlyctenular cases is somewhat over two per cent. Second, phlyctenular disease stands next in importance to ophthalmia neonatorum as a cause of blindness among children. From these two facts alone it would seem that we have here a condition which should be more fully understood than it has been in the past: When we realize, moreover, that children suffering from phlyctenular disease are more than likely to fall victims to tuberculosis later in life, the whole question assumes even greater importance.

Pathology.

Owing to the fact that eyes suffering from phlyctenular disease are seldom excised, at least during the period of the development of the phlyctenule, pathologic studies of the disease focus have been few.

In the recently published atlas of the younger Fuchs of Vienna, there is but one plate illustrating phlyctenular disease; that is, one which shows the breaking down of a phlyctenule into ulceration. Nothing has been added to the previous excellent descriptions by Parsons, Fuchs and others.

The phlyctenule itself appears as a rather dense exudation of fibrin and leucocytes beneath the epithelial layer (either of the cornea or the conjunctiva), with subsequent death of the cells, and circumscribed loss of tissue.

Bacteriologic studies have yielded uniformly negative results: scrapings from the involved areas have shown on direct smear and upon culture no organisms, or at least none which could be held responsible for the pathology present.

For years the possibility of a relationship between general tuberculosis and phlyctenular disease has been hinted at, but such relationship has not been definitely proven, at least by following the recognized methods of research. It would seem that the linking up of these two conditions might be accomplished by extended, careful clinical analysis, rather than by the more definite path through the bacteriologic laboratory, since the latter have yielded negative results.

If a careful study is made of what is now accepted as the probably true genesis of tuberculosis, one finds the following set forth as contributing to the development of that disease: overcrowding, undernourishment, overwork, unsanitary conditions generally: precisely the conditions commonly found in

the homes which furnish us with our cases of phlyctenular disease.

In attacking the problem from the viewpoint suggested i.e., as a broad clinical study, it was found that a rather large unit of workers would be required. In the end, a separate division was established, at the University Eye Clinic, with the aid of the Tuberculosis League of Pittsburgh. The unit was made up of two ophthalmologists, one internist, two nurses, one social worker with two voluntary assistants, and a secretary.

This group met twice a week, when patients were entered and thorough physical examinations made; this included the taking of temperatures and weights, use of fluoroscope, X-ray plates, etc., and the administration of diagnostic doses of tuberculin (in new cases).

The cases were then passed on to the ophthalmologist, for the purpose of having the eye lesion studied, eye conditions noted and local treatment instituted. Forty-eight hours later, the purpose of having the eye lesion studied, eye conditions tions observed and noted. The presence or absence of these reactions were recorded as being *local, focal or constitutional.*

Concerning therapy, this communication is not designed to contribute anything special, excepting in so far as any therapeutic measure might have a bearing upon the disclosure of the causative factor: that is to say, we felt that a definite and continued improvement following the use of tuberculin therapeutically would suggest that this agent possessed some specific curative action, which in itself would be significant.

I am well aware that there exists in the minds of many, a genuine doubt as to the value of tuberculin as a curative agent in tuberculosis in any form. The question as to whether an outbreak of phlyctenulosis necessarily presupposes a tuberculosis infection or not, has not, up to the present time, received a positive answer, this notwithstanding the fact that many observers have given the subject much careful attention.

As indicated above, the ordinary laboratory methods, both in the field of pathology and in that of bacteriology, have brought nothing of value in answer to the question, for the reasons also mentioned above. It may be, however, that sufficient evidence of a more or less circumstantial character can be adduced to be convincing.

In order to reduce the results of our study to a workable basis, a scheme was devised upon which could be entered

those facts in connection with each patient which might be considered as having some bearing upon the possibility of tuberculosis having a causal relationship to the disease.

Attention is here called to chart (A) giving in detail the salient points, viz.

CHART A.

(a) Diagnosis of form of phlyctenular disease.
(b) Age.
(c) Exposure to tuberculosis.
(d) Physical signs other than eye disease.
(e) Tuberculin: treatments, reactions.

Eye record number.	1919. Name	Age.	Diagnosis of ocular disease.	Exposure to tuberculosis.	Physical examination and clinical symptoms.	Tonsillectomy.	Wassermann. +	Wassermann. −	No. of treatments.	Tuberculin. Reactions. Local.	Tuberculin. Reactions. Focal.	Tuberculin. Reactions. Const.	Duration in wks.	Recurrences.
	Buscka, Mary	5	P.K.	.....	Hypertrophied tonsils. Cervical adenitis.				3	2		1	13	Fq.
Y407	Cantoni, Frank	8	P.K.	.....	Cerv. and axillary glands. Temp.	√			1	1	1	1	47	Fq.
16498	Capash, Gertrude	12	P.K.C.	.....	Cerv. glands. Mitral insufficiency. Bronchitis.				35	14	2	1	134	Fq.
W2589	Capazzi, Millie	4	P.C.	.....	No exam. recorded.				..	..	.	..	64	1
	Carlin, Graham	18	P.K.	.....	Cerv. glands. Incipient pulmonary Tb.				1	1			½	
	Carrol, Mrs. Jane	25	P.K.	.....	No exam. recorded.				..	..	.	..	1	
	Charubin, Helen	10	P.K.	Mother died of Tb.	Enlarged tonsils. Slight rt. cerv. gland. Advanced pulmonary Tb.				..	..	.	..	1	Fq.
Y8413	Clayton, Thomas	35	P.K.	.....	Cough. Chest clear.			√	3	1			51	2
Y5471	Cohen, Rebecca	12	P.K.C.	.....	Adenitis. Tonsils removed last year showed tubercles.	√			1	1			1½	Fq.
Y8451	Coltz, Catherine	13	P.C.	.....	No phys. signs. Temp. 99°-99.4°				3	3		1	20	1
Y5955	Conley, Loretta	16	P.K.	Father died of Tb. 6 yrs. ago. Sister.	Cough, Fatigue, shortness of breath, night sweats. Pulmonary Tb. (arrested)	√		√	1	1			2½	Fq.
Y8461	Conroy, Trucella	4	P.K.C.	Father died of Tb.	Enlarged tonsils. Pulmonary Tb. (?)				3	3		2	5	
Y8488	Cretsis, Ecriva	6	P.K.C.	Mother and brother died of Tb.	Tonsils slightly enlarged. Cervical glands.	√		√	18	15		4	110	Fq.
Y5477	Dacy, James	4	P.K.	Cousin in same house	Cough. Cerv. glands.				2	1	1		21	
Y5484	De Riga, Ralph	9	P.K.	.....	Cervical adenitis.			√	1	1			5	

Age.

In order to present the incidence in relation to age, I have prepared a chart (B) which shows graphically how the disease is limited almost entirely to childhood; further, the great prevalence in the third and in the eighth years of life—after the age of twenty, it is practically negligible—while few cases occur until after the first year of life has been completed. (See chart B.)

As indicating the wide differences of opinion between various observers, the following may be noted:

Hamburger states unequivocally that all phlyctenular conditions have a tubercular origin and pronounces them to be conjunctival tuberculides. Von Pirquet declares, without further discussion of the question, for a tubercular origin.

Much states that phlyctenulosis occurs only in the tuberculous. Feer states that the existence of a phlyctenule is certain evidence of tuberculosis of the organism. Axenfeld states that all persons suffering from phlyctenulosis must be considered as tuberculous. Stargardt comes to the conclusion that phlyctenular eye disease, exactly like lichen scrophulosorum and other skin tuberculides, is a genuine tuberculosis.

On the other hand, Czerny considers phlyctenulosis to be the manifestation of exudative diathesis. Moro reports several tuberculosis free, though definitely lymphatic individuals, who suffered from nonspecific face and head eczema, who suffered at the same time from a similar eruption in the conjunctiva. Schelbee makes a similar report concerning a case which received an injection of diphtheria antitoxin, which showed within a few hours the existence of genuine phlyctenules, though the patient was free from any discoverable signs of tuberculosis. Peters declares that according to his view, the etiology of phlyctenulosis is not yet definitely declared, he having found phlyctenules in both trachomatous and diplobacilliary infections. Peters mentions the role played by scrofula, the significance of which condition he thinks is not yet fully understood.

From the above, it will be seen that there is as yet no unanimity of opinion upon this vital point. It would seem that the development of the various methods of inducing the well known tuberculin reaction should throw some light upon the subject, and we think in our present series considerable help can be expected from this source.

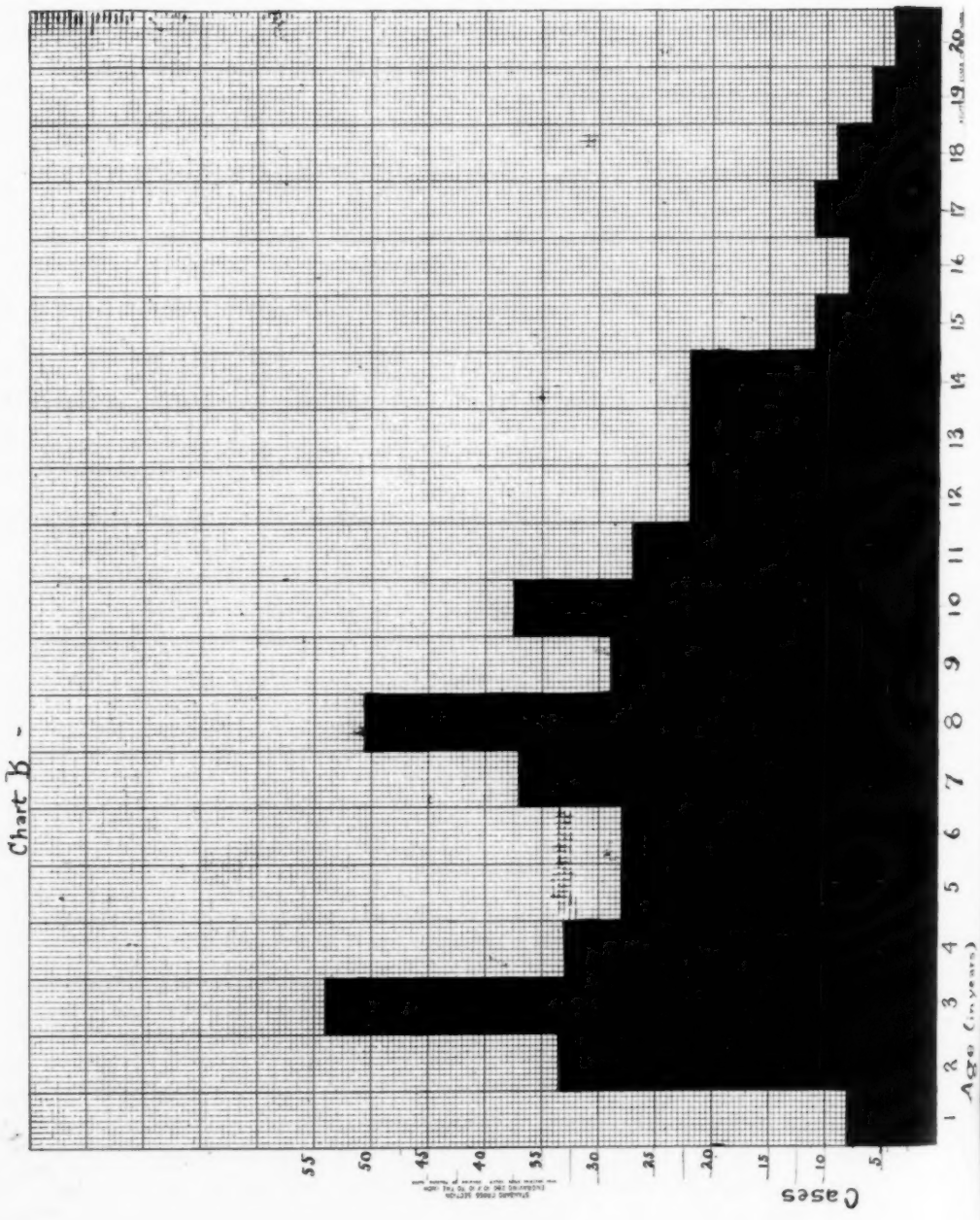


Chart B -
Cases
55
50
45
40
35
30
25
20
15
10
5
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
Age (years)

It is well, in this respect, that certain facts concerning the tuberculin reaction should be clearly understood.

First, in the healthy individual, no effect whatever follows the injection of tuberculin (Igersheimer).

Secondly, the occurrence of a definite reaction following the injection of tuberculin must be taken as evidence, that the subject presenting such a reaction has at some time received a tuberculous infection, though for the time being no active tuberculosis process may be demonstrable in the individual.

Tuberculin reactions occur in three different ways.

First, the reaction shown at the point on the skin or mucous membrane where the tuberculin is introduced—*local reaction.* This appears as an area of redness, slightly elevated, and slightly tender, varying in extent with the amount and strength of the dilution used, as well as the patient's reactionary response.

Second, *focal reaction.* The focal reaction is due to a change occurring in, or in the neighborhood of the tubercular focus, usually consisting of hyperemia, increased secretion, etc.

Third. The *general reaction* consists of a group of characteristic symptoms, chief of which is a rise in temperature, usually accompanied by headache, malaise, loss of appetite, with sometimes nausea and vomiting.

The Vienna school has proved beyond doubt that tuberculin is a trustworthy reagent in the detection of tuberculous infection. In the poorer section of the Vienna population, the cutaneous reaction of von Pirquet, rare in infants up to six months of age, becomes increasingly frequent among older children, and at puberty occurs almost without exception. The postmortem table has shown that this reaction actually coincides with and is an indication of tubercular infection.

In infants, a tubercular infection implies a disease which is nearly always fatal; the infant, if infected, dies from tuberculosis as surely as it dies of smallpox, and treatment is likely to be unavailing. But older children rarely die of tuberculosis, and from the seventh year upward, healed and inactive tubercular glands, combined in most cases with a small healed focus in the lung, are very frequent conditions. It is this condition which gives rise to the cutaneous, as well as to other, tuberculin reactions, which are due to the sensitiveness of the organism produced by tubercular infection. This sensitive-

ness is known to persist after the disease to which the infection gave rise has become quiescent or has been cured.

Illustrating some of these points, I have prepared some charts which give the results of some observers working in the field of *tuberculosis,* in order that we may have clearly before us the commonly found reactions of children, from birth up to the fourteenth year. The figures presented here deal with children who are free from active tuberculosis, in that no clinical signs of tuberculosis were present at the time these reactions were observed. (See charts C, D, E, F, G.)

CHART "C". MULLER.

0 to 3 months 8 per cent reactions
3 to 6 months 7 per cent reactions
6 to 12 months 12 per cent reactions
1 to 2 years 22 per cent reactions
2 to 4 years 31 per cent reactions
4 to 6 years 37 per cent reactions
6 to 10 years 36 per cent reactions
10 to 14 years 54 per cent reactions
Total number of cases=949.

CHART "D". VON PIRQUET.

0 to 3 months 0 per cent reactions
3 to 6 months 5 per cent reactions
6 to 12 months 16 per cent reactions
1 to 2 years 24 per cent reactions
2 to 4 years 37 per cent reactions
4 to 6 years 53 per cent reactions
6 to 10 years 57 per cent reactions
10 to 14 years 68 per cent reactions
Over 14 years 90 per cent reactions
Total number of cases=988.

CHART "E". GANGHOFNER.

Under 3 months 0 per cent reactions
3 to 6 months 5 per cent reactions
6 to 9 months 8 per cent reactions
9 to 12 months 15 per cent reactions
1 to 2 years 12 per cent reactions
2 to 3 years 15 per cent reactions
3 to 4 years 40 per cent reactions
4 to 5 years 36 per cent reactions

5 to 6 years 51 per cent reactions
6 to 7 years 50 per cent reactions
7 to 8 years 50 per cent reactions
8 to 9 years 68 per cent reactions
9 to 10 years 60 per cent reactions
10 to 11 years 58 per cent reactions
11 to 12 years 65 per cent reactions
12 to 13 years 72 per cent reactions
13 to 14 years 80 per cent reactions
Total number of cases=462.

CHART "F"

Bednarski	Positive in 100%
Brenneman and Gibson	Positive in 98%
Rosenhauch	Positive in 96%
Kollner	Positive in 95%
Schreiber	Positive in 93%
Veeder and Hempelman	Positive in 92%
Ayreux	Positive in 89%
Weckers	Positive in 88%
Leber and Hethey	Positive in 75%
Igersheimer	Positive in 70%
Present Series	Positive in 88%

CHART "G". ENGEL AND BAUER.

3 and 4 years 15 per cent reactions
5 and 6 years 16 per cent reactions
7 and 8 years 32 per cent reactions
9 and 10 years 48 per cent reactions
11 and 12 years 36 per cent reactions
13 and 14 years 54 per cent reactions
Total number of cases=288.

Contrasted with these figures, it is significant to observe the resultant reactions where tuberculin was administered to children suffering from phlyctenulosis, but presenting no clinical signs of tuberculous infection. It will be observed that the percentages in this series range from 100%, as found by Bednarski, to 70% by Igersheimer.

Our own series of cases relates to children suffering from phlyctenulosis at the ages of one to fourteen years. The reactions, as shown in the different years, are fairly constant, and give us an average of 88% for the entire period. (Charts I and J.)

CHART "I". PRESENT SERIES.

1 and 2 years 76 per cent reactions
3 and 4 years 81 per cent reactions
5 and 6 years 75 per cent reactions
7 and 8 years 84 per cent reactions
9 and 10 years 77 per cent reactions
11 and 12 years 77 per cent reactions
13 and 14 years 95 per cent reactions
Over 14 years 82 per cent reactions
Total number of cases=305

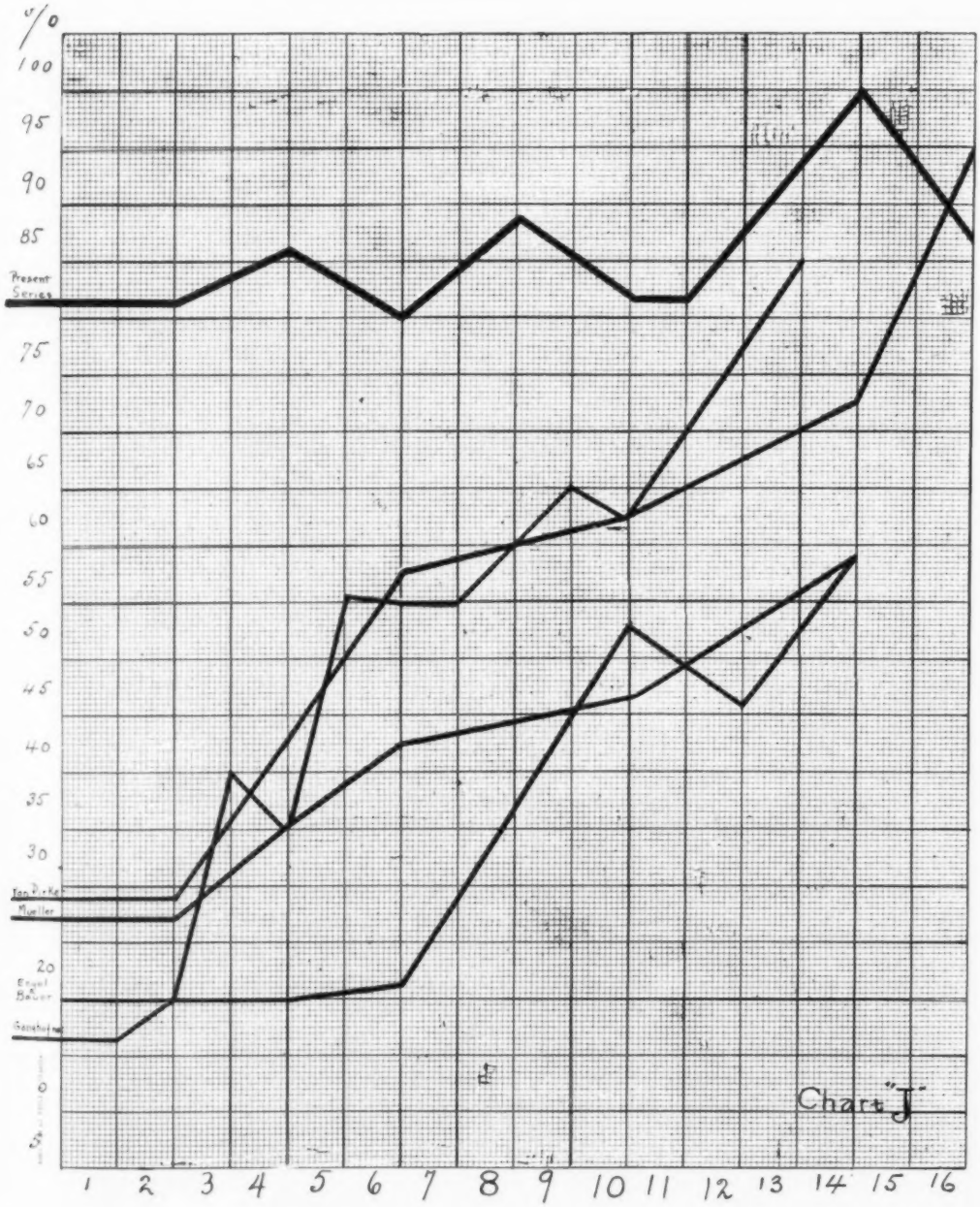
The rather striking comparison between the tuberculin reactions shown by children suffering from phlyctenular disease and those not so afflicted is much more graphically illustrated on Chart "J". This chart presents the findings of von Pirquet, Muller, Engel and Bauer, and of Ganghofner, as compared with our series, which latter group gives the reactions found in children suffering from phlyctenulosis, but presenting no other sign of tuberculosis. The reactions, as shown in the different years are fairly constant and give us an average of 88% for the entire period.

At first glance, it would appear that, if the hypothesis is sound that phlyctenular disease is always an expression of tuberculous infection in the individual, as is maintained by many observers, we should all obtain reaction results comparable to those observed by Bednarski, Brenneman and Gibson, or Rosenhauch. Does the failure to obtain these results vitiate the theory?

The answer to this is that the tuberculin reaction is interfered with and does not obtain in characteristic form when administered to individuals whose health status has been altered by various disease conditions. Igersheimer states that a percutaneous injection will often be followed by a positive reaction, where von Pirquet is negative. It would seem a further and more exact understanding of the conditions under which aberrant reactions may be looked for would throw more definite light on the subject.

CONCLUSION.

While nothing has been brought out here that definitely establishes the supposed relationship between phlyctenular disease and tuberculosis, we are inclined to the belief that the relationship is a very close one, albeit it is a relationship which we cannot invariably demonstrate.


%
100
95
90
85
Present Series
75
70
65
60
55
50
45
40
35
30
Jan. Dec. Ke
Mueller
20
Engel Bauer
Gesetzlose
0
5
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16
Chart "J"

Since we are unable definitely to solve our difficulty by submitting our problem to the laboratory, we are forced to bring in all the circumstantial evidence available, which after all may be sufficient to give us a working hypothesis which shall be satisfactory.

It may be that the efflorescence in the corneal or conjunctival epithelium and subepithelial tissue is simply the response of the organism to the primary infection, which infection seems to be the lot of nearly all members of the human race, at some time during the adolescent or preadolescent period of life.

If this can be demonstrated as a fact, another medicosociologic problem is discernible, in that every case of phlyctenulosis becomes doubly interesting from the standpoint of those who are engaged in the fight against tuberculosis.

In our clinic, we have established a routine which brings to the clinic every member of a household in which phlyctenular disease occurs, each member of such household being then subjected to a thorough physical examination, with the purpose of excluding or discovering the presence of cases of tuberculosis. This, of course, adds to our work, but the results have assuredly more than justified the effort, since we have in this way discovered a number of cases of pulmonary tuberculosis amongst the adult members of a single household —some incipient, some far advanced—usually with no knowledge of the nature of the seriousness of their condition.

This constructive phase of our outpatient department is analogous to our handling of the question of interstitial disease of the cornea—where individual members of each household wherein interstitial keratitis appears are brought to the clinic for examination with the view of excluding or disclosing syphilis by the Wassermann and other tests.

The plan I am convinced is sound and worthy of recommendation, even in spite of our incomplete knowledge of the origin of the phlyctenular disease.

The family history positive for tuberculosis, where father, mother, brother or sister had definite pulmonary lesion, equals 20%.

REFERENCES.

Hamburger. Die Tuberculose des Kindesalter, Vienna, 1913.
von Pirquet. Lehrbuch der Kinderheilkunde.
Much. Kindertuberculose; ihre Erkennung und Behandlung.
Feer. Diagnostic der Kinderkrankheiten, Berlin, 1921.
Axenfeld. Lehrbuch der Augenheilkunde, Jena, 1920.

Stargardt. Bericht der ophthalm. Gesellschaft, Heidelberg, 1916.
Czerny. Jahrbuch der Kinderheilkunde, vol. 61.
Moro. Deutsche med. Wochenschrift, 1909.
Schelbee. Deutsche med. Wochenschrift, 1915, No. 39.
Peters. Handbuch d. allgem. Pathologie und patholog. Anatomie des Kindesalters, Wiesbaden, 1913.
Riviere and Morland. Tuberculin Treatment, Oxford, 1912.
Bednarski. Monatsschrift für Kinderheilkunde, II, p. 855.
Igersheimer. Klinische Monatsblaetter für Augenheilkunde, and Arch. für Ophthalmologie, vol. 76, p. 217.

DISCUSSION.

DR. F. T. TOOKE, Montreal: In the first place, will you be good enough to accept my thanks for your kind invitation to take part in the deliberations of this worthy society; and, in the second place, to accept my apology, if such may be necessary, for the nature of whatever remarks I may have to offer on this most interesting and important subject? That I have not been supplied with the text of the matter in question or even with a summary of what we have listened to, has left me more or less at loose ends as to what my duty should be in entering into this discussion. May I be allowed to congratulate the Academy for having selected so important a subject, and then, to tender my thanks to the author for the profit and pleasure I have derived from listening to a paper which has been so completely prepared and admirably presented?

For it is a matter of no little concern, that so important a subject as phlyctenular keratoconjunctivitis should have been considered as especially worthy of the particular transactions of this society. Any subject which tends to conserve, or preserve, not only the physical, but also the economic forces in any faculty in childhood, is deserving of the most serious consideration on the part of those of us to whom the care of their eyes is entrusted. Preventative medicine has done much to reduce the calamitous loss of vision resulting from gonorrheal ophthalmia; social service has helped greatly in lessening the degree of loss of sight in interstitial keratitis. Phlyctenular conjunctivitis, with its corneal complications and subsequent scar tissue formation, only too often finds a place in the discard as being ordinary and unworthy of any serious consideration. Corneal scars, and thereby a loss of visual acuity, occurs in at least 60 per cent of all cases, as shown in our records at the Ophthalmic Clinic of the Royal Victoria Hospital.

That the subject is eliciting more than a passing attention, in at least one quarter, is vouched for by Sir John Parsons. He told me this summer, that the two very admirable ophthalmic schools at Swanley and Brentwood, organized and administered by the County Council for the poor children of London, had practically been emptied now of all cases of trachoma, the various types of conjunctivitis and marginal blepharitis. The half of one institution was being maintained, and it, for the serious care and consideration of phlyctenulosis in children.

A number of ophthalmologists and pediatricians of undoubted repute would have us believe that phlyctenules are of tuberculous nature. They point to a large percentage of responses of that debatable clinical procedure known as the "von Pirquet reaction method" in this particular class of cases. They also emphasize an associated tuberculous family

history. My query to such a statement is, given an equal number of children suffering from malnutrition, tonsils and adenoids, decayed teeth, bowel infections, starved of fresh air and sunshine, depending upon diseased milk and selected from the same class of society as that in which these children are so frequently found, would not one find a correspondingly high percentage of von Pirquet reactions? My contention is, granted tuberculosis may be an associated condition, pathetically endemic as it is in the children of the slums, is it any more likely to be a predisposing cause than the sources of focal infection to which I have just referred, or any force impairing the development of the child? If we acknowledge the male sex as possessing greater qualities of resistance than the female, it is of interest to note, that in our experience, females form 75 per cent of the cases. Should this necessarily mean that there are then three female tuberculous patients to one male? In the last fifteen cases admitted to the wards of the Royal Victoria Hospital, where most of them were subjected to a routine physical examination, including diagnostic doses of tuberculin and X-ray photographs of the chest, tuberculosis was demonstrated by no means in all the cases. The associated conditions, however, were as follows: 1, Eczema of the lids; 2, Dental caries; 3, Tuberculosis, teeth, strumous rhinitis and tonsils; 4, Tonsils and adenoids; 5, Tonsils, adenoids, strumous rhinitis; 6, Tonsils and adenoids, dental caries; 7, Tonsils and adenoids; 8, Tonsils and adenoids, glands, scrofula; 9, Tuberculosis, scrofula, dental caries; 10, Dental caries; 11, Dental caries; 12, Strumous rhinitis and tonsils; 13, Tonsils and adenoids; 14, Tuberculosis, tonsils, dental caries, Wassermann, 4 plus. May not all these be possible contributing elements inciting inflammatory manifestations in the eye, and of like importance as tuberculosis?

I would like to inquire of the essayist if, in his opinion, the findings of Hayashi and Weekers, and, more recently that of Gibson, to a large measure do not evade the issue? If the absence of actual tubercle structure, the presence of mononuclear leucocytes as well as of polymorphonuclear with definite evidence of mitosis, the absence of characteristic perivascular changes, giant cells, central caseation and tubercle bacilli in the tissues, would not suggest a simple inflammatory nodule rather than a tuberculous one?

Again, in animal inoculation, does he consider that such authors as Bruns, Rosenhock, Rubert and Gibson as well, have, in spite their most painstaking work, proved their point? For so many animals definitely proven to be tuberculous, as in Gibson's series, does it not appear remarkable, that in tuberculin inoculation in the conjunctiva, a definite local reaction was not invariably the case? It was very decidedly the exception rather than the rule.

Until one obtains tubercle bacilli from the scrapings of a fairly representative proportion of these cases, one is not justified in classifying them as tuberculous lesions, though possibly associated lesions with it or some other causes in tuberculosis. This, however, is a horse of another color.

Again, the ready response to treatment, the reestablishment of proper hygiene, fresh air, wholesome clean food and the application of the

recognized local remedies for this particular disorder is altogether out of proportion to the time required to heal ocular lesions definitely established to be tuberculous.

How far indicanuria, through the absorption of indol through the lower bowel, is responsible, as suggested by Signorina Eischnig and Calombo, I am unprepared to state, or the microbic processes of putrefaction found in the lower bowel, or the overindulgence in starches or sugars, which find their way to this part of the digestive tract. Doubtless they are all contributing factors. We do, however, know that indicanuria may be demonstrated in the urine not infrequently in health as well as in disease.

To sum up: Ignorance and the slum, so often found together, are the primary etiologic factors in the production of phlyctenulosis—other factors, of which tuberculosis is only one of many, follow in their wake. If you eradicate these two, the others are to a large measure in consequence deleted.

Dr. Marcus Feingold, New Orleans, Louisiana: We must agree with the essayist and with Dr. Tooke, that the pathology of phlyctenulosis is by no means a simple question. Still, we must not forget one or two reports in the literature, which seem to prove the actual presence of tubercle bacilli in the phlyctenule. My own conception of phlyctenulosis is greatly based on the ideas brought forth by Rubert and published about twelve years ago. His experiments showed, that animals injected with tubercle bacilli or with tuberculin did not develop phlyctenulosis from that treatment alone. Phlyctenules appeared in the eyes of animals so treated only after some form of conjunctivitis or irritation was produced. He also reminds us that diplobacillary conjunctivitis, and different forms of irritation and traumatism are known to produce phlyctenules in individuals so predisposed. It is not the external factor, the conjunctivitis or the traumatism, alone, nor is it the tubercular toxemia alone which produce the phlyctenule, but the combination of the two. This would explain why a phlyctenule, though found in individuals with tuberculosis, seems to be free of tubercle bacilli.

This particular conception of phlyctenulosis appeals to me, because it seems to bear out our own daily clinical experience, and the experience of the country at large. In New Orleans, tuberculosis is much milder than in the north, and is especially milder among the white races. With us, phlyctenulosis in the white race is comparatively rare and nearly always mild. In the negro, on the other hand, who is more subject to tuberculosis, phlyctenulosis is comparatively frequent, and is even quite malignant at times, in the adult. This seems to me to illustrate the dependence of phlyctenulosis on the intensity of the underlying tubercular toxemia. The influence of the provoking factor, on the other hand, is illustrated by the following experiences. In spite of the unusually severe winter of 1923-24, so few cases of acute conjunctivitis were observed in our out-patient service, that no case could be shown to the students. During the same period, only three or four cases of phlyctenulosis were recorded. In contrast with this was the unusual experience of an epidemic of acute monocular conjunctivitis, during the last two weeks of August and the first two weeks of September of this year. Pneumococcus was found in all cases, and all showed enlarged preauricular gland.

About one-half of these cases showed phlyctenules.. Nothing seems to explain these observations as well as the theory that phlyctenulosis is based on tubercular toxemia, and is provoked by some external factor damaging the conjunctiva and cornea.

Dr. Robert Scott Lamb, Washington, D. C.: This is a subject of so much importance, that I think we can afford to take Dr. Tooke's suggestion, and whereas Dr. Feingold may be perfectly correct, I think there are some other factors which, perhaps, he has not considered. One is that negroes, who are more affected in the neighborhood of New Orleans, have a very much more marked tendency to lymphatic development than the whites. Another thing is, that the economic situation has changed there more. They do not see as many phlyctenulosis cases now because the negroes are making much more money than they ever did, and are eating different kind of food.

I think we must consider the metabolic side of this, and I believe if investigation were made of the metabolic rate, it would be discovered that it is low in these cases. The fact is, that some time ago Dr. Bordley brought out the efficacy of thyroid in uveitis. I do not think he went quite far enough in experimental work, because I believe if you increase the metabolic rate with thyroid in small doses, and lactat of calcium about three times per day, you will find these cases will all improve.

ETIOLOGY AND TREATMENT OF SCLERITIS AND EPISCLERITIS*.

WILLIAM L. BENEDICT, M.D.

ROCHESTER, MINN.

Inflammation of the episcleral and scleral coats of the eye results in the loss of many eyes, and leaves a still larger number permanently injured and disfigured. The lighter attacks of the disease may subside without treatment, or with only local treatment, designed to soothe the eye during the most severe symptoms, without leaving a visible mark. However, these attacks usually recur at fairly regular and frequent intervals, and the summation of the combined changes resulting from the attacks will be noted as a discoloration of the anterior segment of the globe. The more severe attacks of inflammation are followed by adhesions of the conjunctiva to the sclera, with consequent irritability of the eyes. There is also a thinning of the scleral coat and of the ciliary body and choroid, often followed by changes in the iris. Recurrent attacks of scleritis so often are experienced by persons who have suffered one severe attack, that a chronic inflammation is produced with exacerbation of severe symptoms. This inflammatory condition may last for years, and when it finally subsides, the eye is disfigured by dark colored patches about the cornea, intercalary staphylomas, staphyloma of the entire anterior segment of the eye, or diffuse cloudy opacities of the cornea. The effects of the disease are familiar to all ophthalmologists. The cause of the disease is too often obscure or only vaguely comprehended. The treatment has been empirical and unsatisfactory. It is my purpose in this discussion to consider the clinical aspects of the disease, to review it with respect to symptoms in other organs and other parts of the body, and to recount my experience in its treatment.

---

*Section on Ophthalmology, Mayo Clinic.

The episcleral tissue is composed of delicate tortuous bundles coursing in various directions. "Outwardly it extends over into the loose tissue which fills out Tenon's space. Toward the inner side its bundles are firmer, thicker, and the mattressing is more close. In this way it graduates over into the tissue of the sclera proper. It is especially characterized by relatively numerous vessels, and so is vastly differentiated from the vesselless tissue which fills out Tenon's space, as well as from the tissue of the sclera, which is scanty in vessels . . . . The vessels in the back part of the episcleral tissue are branches of the posterior ciliary artery; those in the fore part of the tissue, of the anterior. These branches form a network in the usual way; that is to say, one artery is accompanied by two veins. The meshes of this net are very wide in the back part, and become increasingly narrow in front of the recti muscles and toward the borders of the cornea. A thicker capillary and venous net exists only in this anterior zone of the sclera; a marked filling of this net produces the socalled ciliary injection."[3]

"The sclera proper is a dense fibrous tissue in which the bundles cross in varied directions and often divide at sharp angles. The individual bundle consists of fine collagenous fibrillae coursing parallel to each other and to the length of the bundle, ordinary elastic fibers, and others which occupy the periphery of the bundle; only a few extend into the interior between the groups of fibrillae. As a whole, the elastic fibers course parallel to the direction of the bundles, and are much more delicate than in other tissues. Between the bundles, especially in the angles formed by this crossing of each other, lie the fixed cells of the scleral tissue. The blood vessels of the sclera fall into two groups. The one makes use of the sclera only for passage, and subdivides into capillaries in other tissues; the vessels of the uvea belong in this group. . . . There are, however, a large number of smaller vessels of the sclera, which do not properly supply this membrane itself, e.g. in the neighborhood of the optic nerve. Moreover, in the most anterior portion of the sclera, just behind and outside Schlemm's canal, relatively numerous vessels are present, for the anterior ciliary veins pass through here. How insignificant the second group is, i.e. how few vessels the sclera possesses in and of itself, comes out best in the region of the equator; here and there a capillary lumen is visible, but

in a cursory inspection this tissue appears entirely devoid of vessels."[6]

If neoplasms and diseases of the subconjunctival tissues that arise from external sources are omitted from consideration, there still remain blood borne inflammatory lesions, both acute and chronic, having their origin in some remote part of the body. The principal distinguishing feature of the inflammation in the eye is the color during the congestive state, which in turn is determined by the depth of the dilated vessels beneath the conjunctiva. The characteristic purplish hue is caused by congestion in the network of the anterior ciliary vessels. This color does not disappear when the eye is pressed on, although the congestion of the conjunctival vessels, which always accompanies the congestion of the ciliary vessels, disappears as long as pressure is applied. There is only moderate edema of the conjunctiva and episclera, even with severe inflammation of the sclera. Severe chemosis indicates a more superficial localization of the disease. Tenderness on palpation is often a prominent symptom. Pain on rotation of the eyes is not characteristic of scleritis; it usually indicates that the disease is located in the globe behind the line of insertion of the recti tendons or, more frequently, in Tenon's capsule. In turn, scleritic and chronic episcleritic induration and congestion are most prominently displayed, either as small nodules situated usually between the equator of the globe and the limbus conjunctivae, or as broad, indurated swellings that may appear to cover less than one-quarter of the anterior sclera, or may entirely encircle the cornea (annular scleritis). The latter form is usually seen in the chronic cases after many attacks localized in a smaller portion of the area, the adjacent uninvolved areas of one attack becoming involved in the subsequent ones.

In eyes in which the cornea is almost entirely encircled by the brawny swelling, the cornea becomes steamy, due to infiltration of the deep and superficial layers near the limbus, and the more superficial layers near the center, the white marks seen in the cornea assuming the form of interlocking rings or coalescing clouds, characteristic of sclerosing keratitis.

The structure of the episclera fades almost imperceptibly into the capsule of Tenon on one side and into the sclera on the other. Inflammation of any one of these structures must

necessarily involve the tissue adjacent, yet so different is their course and significance that "tenonitis," "episcleritis" and "scleritis" are recognized as clinical entities. The clinical pictures of these conditions are adequately described in most text books on ophthalmology, and are quite generally accepted. Each has also its subdivision, based on some characteristic of its presumed etiology or its course, such as "syphilitic", "fugacious," "persistent," "deep," and "annular." Parsons says, "It is impossible, pathologically, to draw any hard and fast line between the deeper forms of conjunctivitis, or episcleritis, and true inflammation of the sclerotic itself, or scleritis. In the very limited number of cases which have been examined microscopically, the general type of inflammation has been characteristic, but the relative extent has varied considerably and apparently out of all proportion to the clinical severity. . . . . Even clinically, episcleritis merges into scleritis, and the latter, though usually nodular like the former, is invariably more diffuse, and in rare cases invades the circumcorneal area."[1]

"Uveitis is a common complication in episcleritis, and almost always occurs with the brawny type of scleritis, although whether as a primary or secondary affection cannot be determined. There are reasons for thinking that they are fundamentally due to one and the same cause, although the lesions may not correspond in location. The sclerotic nodules show a predisposition to attack the neighborhood of the anterior perforating ciliary vessels, and extension of the inflammation is usually along them. In many cases, the inflammation is equally marked in the sclerotic and in the uveal tract. In these it is impossible to draw any deductions as to priority of onset".[2] "Tuberculosis is often mentioned as a probable factor in the production of scleritis, but of the eyes examined, only a few have shown the presence of tubercle bacilli, and the accompanying giant cells found are often designated as ordinary foreign body giant cells (Fremdkörper-Riesenzellen). Gout is given as a predisposing cause, particularly in old persons with the brawny type of sclerosis. Rheumatism is mentioned in all textbooks in connection with the etiologic factors in scleritis, but without referring to the cause and nature of the rheumatism, we are far afield in our conception of the real nature of the disease. In many reports of cases of scleritis, rheumatism is specifically stated to be absent, and no specific cause of the disease is mentioned. Parsons believes that "The

extreme chronicity of all forms of the disease points to a specific inflammatory process, a view which is borne out by the nature of the leucocytes present. The advance is doubtless impeded by the extreme resistance of scleral tissue to invasions of all kinds, inflammatory or neoplastic; and perhaps, also, by its direction contrary to the normal lymph flow. The line of exit of the venae vorticosae seems to limit the backward progress of the inflammation, and this is possibly due to better nutrition of the sclerotic behind the equator."[3]

The fugacious forms of scleritis are said to be brought on by eyestrain, exposure to inclement weather, use of the eyes in very bright light, disturbances of digestion, rheumatism and gout, and the chronic forms are due to rheumatism and gout, and probably digestive disturbances. Another author believes that the disease occurs more frequently in women than in men, the attacks in women often coinciding with the period of menstruation, and being more frequent among the old than the young. It has been shown quite conclusively, in recent years, that rheumatism and arthritis, as well as non-suppurative inflammatory lesions throughout the body, are due in many cases to the presence in the blood stream of bacteria (streptococci), or toxins produced by such bacteria when localized in some remote organ or focus of infection. The fact that young persons should be less susceptible to attacks of inflammation, is in keeping with their more vigorous resistance to infection and the better condition of such organs as usually harbor bacteria in older persons. Furthermore, the site of inflammation in rheumatism is usually around joints, or at the juncture of muscle and tendon, and in periosteum, where there is a change from a highly vascularized to a poorly vascularized tissue, with resulting diminution of available oxygen, and consequently diminished resistance in invading organisms.

The character of the tissue involved and its peculiar blood supply, as noted, form the groundwork for the production of focal infection lesions in the sclera. In the subconjunctival and choroid tissues are beds of capillaries and small arterioles capable of becoming congested and providing a rich supply of blood, while the sclera, itself, containing only small vessels for its own nourishment, cannot be supplied with sufficient blood to give a proper resistance to bacteria or to their toxins. Hence, the smaller vessels in the sclera and the emissary veins

in the ciliary portion provide local points of diminished resistance and favorable location for chronic infectious processes.

"From the microscopic study of sections of muscles in chronic myositis and arthritis deformans and of those in animals following repeated injections of these strains, a most interesting mechanism in the production of the lesions has been discovered. Evidence has been obtained as to why cure in these chronic affections is so difficult, and the reason massage and applications of heat are such valuable agents in their treatment. The reaction in these very chronic conditions is not leucocytic, but mainly mononuclear and endothelial. The endothelial cells lining the small blood vessels become extremely swollen, proliferate, and, in consequence, the lumen of vessels, including arterioles, becomes partially or completely obstructed. The supply of available oxygen is thus lowered and the growth of the organisms favored, for it has been found that the streptococci from the lesions in muscles are very sensitive to oxygen. The common occurrence of spontaneous and experimental lesions in flat muscles, or in the tendinous ends where the normal blood supply, and, consequently, the supply of available oxygen in response to insult, are relatively low, would seem to be due to the same cause."[4]

The search for foci of infection in patients with scleritis must, in the present state of our knowledge, be conducted on fairly general lines, and any focus found considered a probable source of the disease. Peridental infections are the most common. Experimental studies have led to the conviction that a large number of diseases of the eye are due to the spread of organisms from abscesses around the roots of teeth. Iritis, cyclitis and choroiditis have been produced in animals by injection of bacteria grown from peridental abscesses of persons having acute attacks of these diseases. Bacteria from the tonsils, paranasal sinuses, infected gall bladder, and genitourinary tract must be looked for, and some thought given to intestinal intoxication. The increased frequency of the disease in women and the coincidence of an exacerbation of symptoms with the menstrual period, led me to obtain cultures from the cervix uteri in cases of both acute and chronic scleritis. Vaccines made from these cultures were administered with most gratifying results.

Report of a Case.

Case A365928. Miss J., an art teacher, aged twenty-six

years, came to the Clinic July 25, 1921 because of chronic inflammation of the left eye. The right eye had been divergent since childhood. Vision of the right eye was 6/6, of the left 6/15. Seven months previously, both eyes, which until then had been free from disease, became painful and congested. The patient consulted an oculist who made a diagnosis of episcleritis. A general physical examination was negative. Examination of the teeth and throat did not reveal any focus of infection. Salicylates were given, and after two months the eyes became clear. Five months later, the right eye became inflamed again, and salicylates were again administered, this time without benefit. The upper half of the anterior sclera was edematous, injected, purple, and tender to pressure. The episcleral and scleral coats were involved in the inflammation, the cornea remaining clear. After three weeks of local treatment, during which time the condition had improved, the symptoms became distinctly more severe and the vision blurred. The Wassermann and von Pirquet reactions were negative. It was then ascertained that at each menstrual period the eye became more inflamed. Further pelvic examination revealed no lesions, but cultures were made from the apparently healthy cervix uteri. Short chain, hemolytic streptococci were grown in pure culture and a vaccine made from it, which was administered at weekly intervals with immediate improvement in the condition of the eye. Within four weeks the inflammation had subsided, and no further trouble has occurred.

Since then, three other persons having recurrent episcleritis have been treated with autogenous vaccines with equally good results.

Several persons have had attacks of episcleritis followed by the frequent occurrence of phlyctenulosis. While the eyes bore the slate gray discoloration of deep scleritis, the conjunctiva contained typical nodular phlyctenules near the cornea, without signs or symptoms characteristic of inflammation of the deeper tissues. Further search for foci of infection in such cases revealed devitalized teeth or persistent infection in other organs, such as the prostate or cervix, that had failed to clear up under treatment. Eradication of these foci, with local administration of adrenalin solution and calomel powder, caused the phlyctenules to disappear. None of the patients treated with autogenous vaccines had phlyctenulosis.

The fact that scleritis is not a disease of old age was demonstrated by the patients observed. In the Mayo Clinic, during the last two years, the average age at onset was less than thirty-three years; the youngest patient was twenty, the oldest fifty-four. They were nearly equally divided between the sexes. Only one patient reacted to a positive tuberculin test. All had a definite focus of infection. Three patients with recurrent deep scleritis have been treated with tuberculin without noticeable improvement. Only one gave a history of general tuberculosis, a young man who had tuberculous glands, and whose scleritis began at twenty-one years of age. A history of "rheumatism" was not obtained, although localized arthritis and muscular pains were occasional complaints.

Treatment.

Local treatment should be commenced as soon as possible. The pupils should be dilated and the eyes protected from excessive light by dark glasses. Instillation of adrenalin solution, 1 to 1,000, every three or four hours will often bring about a marked diminution of the edema and congestion of the tissues, and will relieve pain in the eye. The application of the electric thermophore at 140 degrees for one minute in direct contact with the small nodular forms of the disease, greatly hastens their disappearance.

Persons with acutely inflamed, severely painful eyes should be hospitalized for general treatment. In my experience, parenteral injections of boiled milk have given the most satisfaction. Four injections of from 5 c.c. to 20 c.c. is usually sufficient. Immediate search for infection about the teeth, tonsils, sinuses and pelvic organs should be thoroughly carried out, and later, studies of the gall bladder, appendix, and kidney made. All questionable foci, such as devitalized teeth and small cryptic tonsils, should be removed, and pelvic lesions appropriately treated as long as any sign of disease persists. Disturbances due to focal infection usually break out anew at irregular intervals unless the original source of infection can be eradicated. Resistance of such recurrences can best be maintained by using autogenous vaccines with salicylates, mercury or iodids internally, and local instillation of a solution of zinc sulphat, 1 gr., and adrenalin solution, 15 minims to the ounce, into the eye three times daily. Warm, moist compresses add materially to the comfort of the patient during the acute stages of the disease.

Conclusions.

1. Scleritis and episcleritis are fundamentally similar diseases brought on by the same causes.

2. The most common cause of scleritis and episcleritis not due to external agents is focal infection.

3. The most common sources of such infection are diseased teeth, tonsils, and pelvic organs.

4. Removal of all known sources of infection is advisable when it will not result in serious danger to the well being or life of the patient.

5. Recurrent episcleritis in women, with exacerbation at the menstrual period, are often caused by pathogenic bacteria about the cervix. Autogenous vaccines made from bacteria grown from lesions about the teeth or cervix have given the best results of any treatment.

6. If severe pain demands immediate treatment, the parenteral injection of boiled milk is of great value.

BIBLIOGRAPHY.

1. Parsons, J. H. The Pathology of the Eye. New York, Putnam's Sons, 1904-1908, i, Pt. i, p. 270.
2. Parsons, J. H. The Pathology of the Eye. New York, Putnam's Sons, 1904-1908, i, Pt. i, p. 272.
3. Parsons, J. H. The Pathology of the Eye. New York, Putnam's Sons, 1904-1908, i, Pt. i, p. 276.
4. Rosenow, E. C., and Ashby, Winifred. Focal Infection and Elective Localization in the Etiology of Myositis. Arch. Int. Med., 1921, XXVIII, pp. 274-311.
5. Salzmann, M. The Anatomy and Histology of the Human Eyeball in the Normal State, etc. Transl. by E. V. L. Brown, Chicago, 1912, pp. 21-22.
6. Salzmann, M. The Anatomy and Histology of the Human Eyeball in the Normal State, etc. Transl. by E. V. L. Brown, Chicago, 1912, pp. 24-25.

DISCUSSION.

Dr. R. A. Kerry, Montreal: May I, first of all, very briefly express my thanks to the officers and Fellows of the Academy for their hospitality and my deep appreciation of their kindly courtesy. In common, I think, with everybody who has attended these meetings, I have derived a great deal of profit and no little pleasure from them.

Dr. Benedict's very interesting paper raises so many points for discussion, that it is practically impossible to touch upon more than a few of them. It refers to the fact that the infection is probably carried by the vessels, but it is significant that foci of scleritis do not occur in the pericorneal area of vascularization, but farther away from the limbus, where the tissue is probably not so well nourished. If one looks for purely anatomic reasons why the disease does not extend backward, presumably the fairly abundant lymph supply found in the neighborhood

of the venae vorticosae afford some explanation, while the intracapsular ligament, or collarette of Motais, which runs around the equator of the eyeball, interposes an effective mechanical barrier and shuts off the posterior segment.

The work of Carrel and his associates, as well as our own clinical experience, teaches us that serum is not an ideal culture medium, a point which has some bearing on the discussion on infection in cataract yesterday. It would seem reasonable to suppose that the location of foci in scleritis is determined by the fact, that they are situated in a poorly nourished section of the eyeball. Still, we must recognize the fact that there is no disposition on the part of foci to spread. They tend to disappear, and fresh foci to break out in another location in the same band of tissue.

As to the etiology, so many observers of authority have told us that tuberculosis is the most common cause of scleritis, that I think we must take this statement as well established. The simple fact, that in doubtful cases bacilli have not been demonstrated, is merely negative evidence, and Groenouw cites an instance in which over 120 sections had to be made before bacilli could be found, and states that quite frequently 40 or 50 sections have been made before bacilli could be found. In addition, a number of observers have pointed out that tissues in which bacilli cannot be demonstrated, but which have been used for inoculation experiments, have produced active disease. They incline to the opinion, that although bacilli cannot be found, the spores are present in the tissues.

As regards the type of disease, I think that both the fibrous and the more destructive type are found in scleritis, as well as in tuberculosis elsewhere. The cause of this variation in type is not known. Hill, who has had a good deal of sanitarium experience in England, seems inclined to claim that the destructive type is due to humanized virus, while the milder cases are due to the bovine type, but as his destructive cases were nearly all pulmonic and likely to be mixed infection, and his others were bone lesions, the statement must be received with some doubt. The fact that we are dealing with two variable factors, variation in the type of germ and in the resistance of tissues to the infection, means that we must expect a marked variation in the result.

Admitting that scleritis is very frequently tuberculous, the number of septic cases reported during the past few years proves that the disease is also at times a manifestation of septic infection. Oddly enough, one finds very little mention of the streptococcus as a cause in the literature, while the staphylococcus has been much more frequently found, one observer reporting eighteen cases. There is evidently need for much research on the question of etiology in this disease.

Dr. Benedict's treatment interests me very much indeed. We have somehow gotten the idea that autogenous vaccines have not proved uniformly successful. In an extremely interesting paper, read here some years ago by Dr. Price, of the Research Laboratory of the American Dental Association, it was pointed out that germs removed from the host, planted on entirely different media, grown under different conditions, and subcultured once or twice, probably changed their properties and affinities altogether; so that the effect of their use as vaccine was quite problematic.

In any case, it is doubtful whether germs grown under such circumstances would develop much toxin which would be active in the body.

Some time ago, I had an atypical case of scleritis, which was very resistant to treatment. Tubercle bacilli were found in the urine, and upon treatment being directed to the vesical trouble, the eye condition cleared up in a very few days. A year or so afterwards, two precisely similar cases were reported in the British Medical Journal. It seems to me, that if distant local treatment were used in Dr. Benedict's cases, the result might in a large measure be due to it.

My own cases have been treated by trying to raise the resistance of the body, and for this purpose we have been using hypodermic injections of iodin (normally excreted by the leucocytes in their defensible warfare), with very satisfactory results. After the close of the War, finding it was practically impossible to get pure oil, I turned my attention to colloid iodin, and finding a preparation on the market of a strength of 1 to 2,000, which I judged entirely too weak, made one forty times as strong, i. e., one in fifty, and having tested it on myself, have used it with very satisfactory results. That the leucocytosis caused by iodin is an active one, is evident to anyone who has seen a single dose of one-quarter grain pull an hypopyon to pieces, or open the eyes of a child with severe blepharospasm.

If we are to handle our cases of infection effectually, we must broaden our base of attack by great extension of our biologic studies. Of this method, Dr. Benedict's paper furnishes a most admirable example.

Dr. E. L. Jones, Cumberland, Md.: It is very nice, if time and opportunity permit, to search for the cause of focal infection, but I wish to speak of a thing that I have used from time to time in these old chronic cases of months' standing, when they come in with infiltration all around the cornea. In addition to giving them whatever constitutional treatment you might see fit, and diet regulations, I use multiple cauterization with an actual cautery. If the inflamed tissue is all around the cornea, I then give a massive injection of mercury cyanid, and it is astonishing how quickly they get relief. We must set up a physiologic reaction in excess of the pathologic, and by this burning and injection we set up an immense physiologic reaction. Those who have not used this treatment will be surprised to see how rapidly the cases improve. Then, after they improve, you can try to find out the pathologic cause.

SENSITIVE TEST CHARTS FOR REFRACTION.*

JONAS S. FRIEDENWALD, M. D.

BALTIMORE, MD.

When the determination of errors of refraction and their correction by glasses was put on a sound scientific basis by the monumental work of Helmholtz and Donders, a variety of test charts were at first advanced by various authors; but among these, the charts devised by Snellen soon took precedence. The principles and general form of these charts have been so universally adopted, that there is at the present time little essential difference between various test letter charts in common use—so little difference, in fact, that a practicing ophthalmologist could easily replace his chart by some one of the other varieties without producing any significant change in his office routine. Such widespread uniformity of

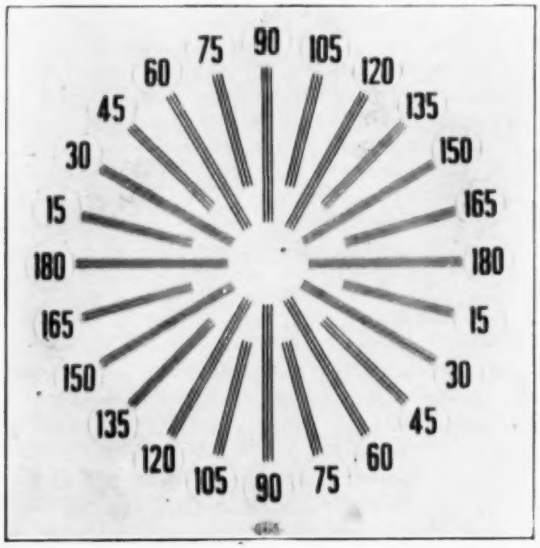


Fig. 1. Wallace astigmatic dial as seen by an eye with 0.25 D. myopic astigmatism. Note the grayness of the blurred lines.

*These charts can be obtained from Bowen & King, Opticians, 405 N. Charles St., Baltimore, Md.

test letter charts attests clearly to an equally widespread satisfaction with these charts. They have been tried and found useful.

The test charts of Snellen and their various modifications were devised to serve two fundamentally distinct purposes: first, to measure visual acuity; second, to determine the error of refraction. While both these purposes are served in a more or less satisfactory manner by the usual charts, it would seem possible to devise a chart more satisfactory for one of these purposes by devoting one's attention to this purpose alone. The charts which are now to be presented are an aim in this direction. They can, in no sense, supersede the Snellen

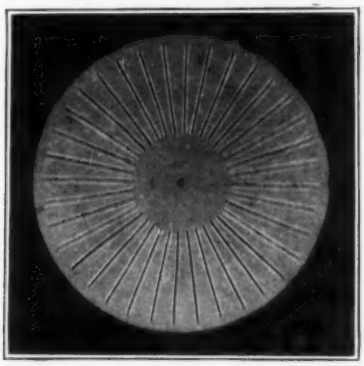


Fig. 2. Author's dial photographed under same conditions as Fig. 1.

charts. They are simply an adjunct to the other charts, to be used not in the measuring of acuity but in the more accurate determination of refractive errors. They are, I believe, more sensitive to small errors of refraction than are the usual charts, or to put it another way, their visibility suffers more in small refractive errors than does that of the other charts.

The principle on which this chart is based is a natural extension of that used in the dials for the determination of astigmatism, which I have published elsewhere[1]. It may briefly be recapitulated here. When a black line on a white background is viewed by an eye with a refractive error, every point in the surface is represented by a diffusion area in the image, and the diffusion areas of adjacent points overlap. The diffusion areas of the line will, then, overlap with that of the background, and depending on the degree of the blurring, the line will appear less and less black, lighter and lighter gray,

until, with a sufficient error of refraction, it can no longer be distinguished. If, now, we construct a chart not simply black and white, but one in which the elements are black with a white border on a gray background, then with only a slight

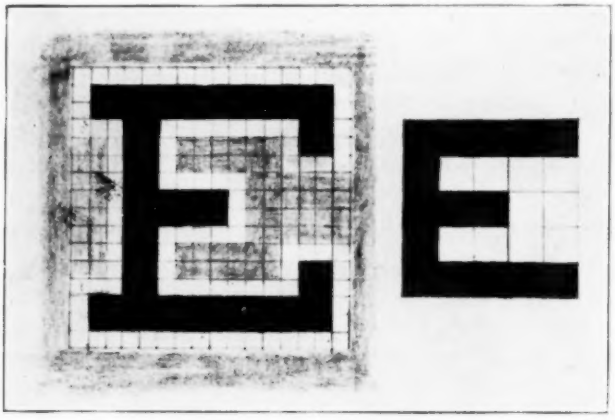


Fig. 3. Construction of the author's test letters compared with those of Snellen.

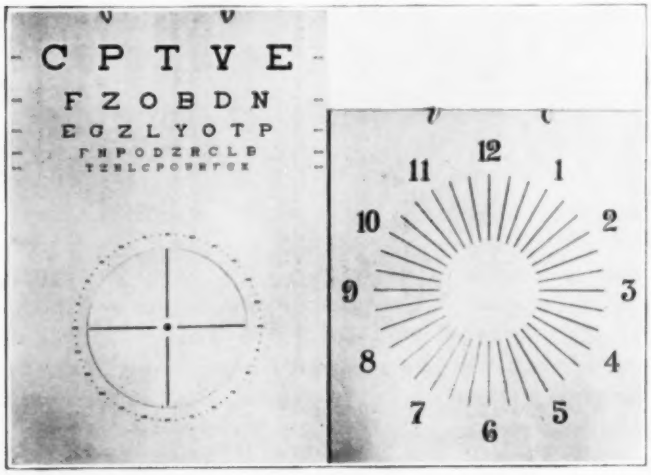


Fig. 4. Set of author's sensitive test charts.

error of refraction, the black and white become fused into a gray, to be completely lost in the general gray of the background.

The Snellen letters, as is well known, are so constructed that the height of each letter is five times the width of the

bars of which the letter is composed. In order to allow for a white border, the letters in our chart have been constructed not on a five to one, but on a seven to one ratio, as is shown in the illustration. The size of the letters is such that the width of the black strokes in the letters subtends one minute of an arc, and the height of the letters seven minutes of an arc at sixty, forty, thirty, twenty and fifteen feet. The lines

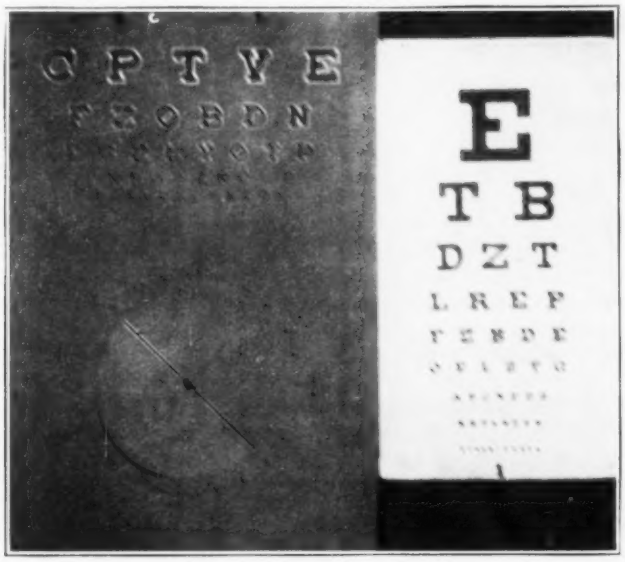


Fig. 5. Comparison of the effect of 0.50 D. myopic astigmatism on the sensitive test chart and on the Snellen test letter chart.

of letters are numbered accordingly, but this must not be taken to mean that if the patient reads the twenty foot line at a distance of twenty feet, we can write down his visual acuity as 20/20, for the visibility of this chart is not directly comparable with that of the Snellen charts.

For the sake of convenience the test letter charts have been printed together with the astigmatic dials already described. The photographs illustrate the appearance of the charts when viewed through various refractive errors.

BIBLIOGRAPHY.

1. American Journal of Ophthalmology, 1924, No. 1.

DISCUSSION.

DR. JOHN GREEN, JR., St. Louis: With Doctor Friedenwald's permission, I should like to refer to some points in his first paper, "A New Astigmatic Chart" (Am. Jour. Ophth., Series 3, Vol. VII, No. 1, p. 8), to which the paper presented today is supplementary.

Before the invention of the ophthalmometer and the development of the shadow test to a point of practical usefulness, the Snellen test letters and astigmatic charts were our main reliance in determining the degree and axis of the cylindrical component of the spectacle lens. The test letters proved reasonably satisfactory in the determination of astigmatism, as well as axial ametropia, and even today are accepted and used by most refractionists. On the other hand, the great variety of astigmatic charts, as Doctor Friedenwald well says, "bears excellent testimony to the very general lack of satisfaction obtained from any of them."

In this connection, it may interest you to learn that my father, who devised a number of astigmatic charts upon which he relied in his early professional career, rarely resorted to them in the last twenty years of his practice. It is my impression, too, that the majority of contemporary colleagues find astigmatic charts useless, and hence do not include them in their equipment for refraction.

While freely acknowledging the valuable information obtained objectively through the use of the ophthalmometer and refined methods of skiascopy (especially skiascopy with cylinders), and subjectively with the aid of the cross cylinder (method of Jackson and Crisp), I am firmly of the opinion that a sensitive astigmatic chart is an essential part of the equipment of one who aspires to the highest possible accuracy in the measurement of astigmatism. Devotees of the shadow test are far too inclined to accept as final the astigmatic correction indicated, if only the spherocylindrical combination thus revealed yields 6/6 vision. Many such "corrections" would by no means stand the acid test of the charts of Lancaster or Friedenwald.

The need for greater sensitivity in an astigmatic chart was partially met by Verhoeff, when he published his radii crossed by concentric circles. Lancaster, insisting on the greatest possible contrast between the blackness of the lines and the whiteness of the background, devised his chart of velvet ribbon radii glued to the whitest of "wedding stock." I have had these charts in daily use for the past eight years, and they have proved eminently satisfactory. In dealing with low astigmatism, the patient often finds difficulty in differentiating between four or five adjacent radii. The sensitivity of the chart may be increased (as I pointed out in discussing Doctor Heitger's paper "The Routine Use of Minus Cylinders in Refraction," Trans. Sec. on Ophthalmology, A. M. A., 1922) by beginning the search for the precise axis with the astigmatic chart in darkness, and gradually increasing the illumination until one or two radii "develop."

I can confirm Doctor Friedenwald's observation concerning the surprising frequency of slight irregular astigmatism associated with the regular type. The appearances illustrated by Doctor Friedenwald in his first paper, I have frequently noted in testing patients with Lancaster's chart under low illumination.

The advantage of a wide pupil in making the patient more sensible to his own astigmatism has been mentioned by Doctor Friedenwald, who suggests eucatropin for this purpose in refracting presbyopes. But why not add to the advantages of the mydriatic those of the cycloplegic? It has been my custom to refract 95 per cent of all patients between 40 and 50, and over 50 per cent of those between 50 and 60, with homatropin. Use cautiously, under the immediate observation of the oculist, with the mydriasis counteracted with eserin and pilocarpin immediately at the conclusion of the test, it has proved harmless and eminently satisfactory in enabling me to determine the astigmatism with accuracy.

I must take issue with Doctor Friedenwald's statement, that in case of low visual acuity, the accurate determination of the astigmatism is not important. In my experience, it is in just these amblyopic eyes, especially if the amblyopia be unilateral, that the most exact correction of a compound error will often add greatly to the patient's comfort and may, in the long run, increase his acuity.

Has Doctor Friedenwald tried a background of darker grey than No. 5 Herring—such a grey as would result from revolving a disc consisting of a white sector, one-third, and a black sector, two-thirds of the whole disc? Would not such a shade more nearly approximate the shade produced by the overlapping of the diffusion areas of the images of the white bordered black radii adjacent to the line representing the true axis? Would not this device still further heighten the sensitivity of the chart, by increasing the contrast between the one selected radius and the radii immediately adjacent?

Doctor Friedenwald's charts reached my office only the day before I left St. Louis, so that I had little opportunity to test them out. Trying them "on the dog" (myself) with +.25 sphere in the trial frame, I found no difficulty in selecting as best the 110° and 120° lines. The cross lines were equalized by —.25 axis 25°. Observing the lowest line of letters (with +.25 axis 115°) they began to fade when the cylinder was swung 7° either way from 115°. Viewing the 6/5 line of my Thorington test card, no change was noted in distinctness of the letters until the cylinder had been swung 12° either way from 115°. In a patient who showed +1.5 sphere $\bigcirc$ +1 axis 90°, a swing of the cylinder to 87° was productive of easily appreciable blur of Friedenwald's smallest letters.

In using astigmatic charts of any sort, it is essential that the examiner be thoroughly en rapport with his patient. The latter must be taught the meaning of the test, and the examiner must not rest content until he is convinced that the lesson has been learned. As there are "born" teachers, so there are "born" refractionists, and much the same qualities of mind and heart are needed by both.

Doctor Friedenwald deserves the thanks of ophthalmologists for his clever utilization of well known optical principles in devising test letters and astigmatic charts which will, in my opinion, insure greater accuracy than has hitherto been possible.

Dr. Jonas S. Friedenwald, Baltimore, Md. (closing): Doctor Green has brought up two points that I wish to comment upon. In my previous paper, I stated that the special purpose in developing these charts was to aid in refraction of individuals whose visual acuity was high and astigmatic error slight. I think these are the cases which present the

greatest difficulty in refraction, and it was for that reason that I placed so much emphasis upon this particular group of cases, and laid aside the cases with larger errors of refraction and less visual acuity. Of course, I agree that accurate refraction is desirable in those cases also, but a large refractive error is not so difficult to correct as a smaller one. The usual charts with heavier black lines are more serviceable in the refraction of patients with less visual acuity than charts with finer lines.

The second point is the question of using darker gray. I tried grays of various tints, and I believe the darker gray is not so satisfactory. It is true, that with a darker gray background, the lines near the axis of astigmatism are even harder to see than are the same lines on the chart with the lighter gray background. One might expect, then, that the sensitivity of the chart would be increased by darkening the background, but this is not true, for as soon as we darken the background, we decrease the visibility of even the best lines, and the patient then finds great difficulty in picking out any line as the best.

TRANSACTIONS

OF THE

TWENTY-NINTH ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

OTO-LARYNGOLOGIC DIVISION

MEDICAL REMEDIES FOR THE CONTROL OF HEMORRHAGE.

SAMUEL G. HIGGINS, B.S., M.D.

MILWAUKEE, WIS.

The immediate postoperative concern of the oto-laryngologist centers on hemorrhage. Any contribution that may lessen this burden, I feel will be accepted. My purpose will be to suggest for your discussion those medical remedies that we have all used, and to contribute my verification of the pharmacologic action of one important official drug that has not as yet been generally used by men of our specialty. The experiences of the various members of this society will add more to clinical information than I could present today by a review of the laboratory findings of each drug. I have found no medical means superseding the fundamentals of exact surgical principles. Surgical technic with regard to the use of ligatures, where indicated, properly applied dressings, and technic in septic areas may not be disregarded in the hope of finding extraordinary aid by known medicinal remedies.

The management of the patient I consider important. This includes the deportment of both the patient and the surgeon. The surgeon should interest himself in the reasonable expectation of the results of the contemplated operation, not the adjustment of an anomaly. The history may contribute much to the knowledge of the pathology of the case. I know of no medical remedies that can be depended upon to control true hemophilia. The assistants and equipment at hand may determine the choice of place for operation—the patient's home, the surgeon's office, or the hospital. As a rule, I have not found that patients with postoperative bleeding do as well at home as in the hospital. That the surgeon's control of his patient is more than psychic, is shown by less hemorrhage in those patients to whom your instructions were specific. This includes our study of the requirements of each case, and this in turn inspires confidence, obedience and acquiescence.

Your experience will suggest the remedies you have used to

avoid excitement and fear. Sedatives may be properly used before and after operations. The bromides or such remedies as sulfonal have a place. Many patients remain quiet and are permitted to remain undisturbed following hypodermic injections of morphin gr. 1/8, hyoscin gr. 1/200, two hours preceding, one hour preceding, and at the time of applying the local anesthetic. The patient's idosyncrasies to those drugs have an important bearing upon postoperative hemorrhage. Should one always give morphin in event of hemorrhage, is a question to be determined by the surgeon.

The choice of anesthetic is influenced greatly by the effect upon bleeding. The increased vascularity of the etherized patient's head usually excludes ether for intranasal surgery. The local action of ether is not the same. I have occasionally directed the anesthetist to drop a few drops of ether in the mastoid wound, which quickly evaporated with some styptic effect. Chloroform produces less cranial congestion. Some of us have seen the sudden pallor and appreciate its dangers. The cyanosis causes us also to forget that it may, in some instances, be legitimately employed. The shrinking effect of cocain, accentuated with adrenalin, is the choice for intranasal surgery. The superficial hyperemia induced by rubbing cocain paste on the septum does not favor a dry operative field. The anesthetic effect may be as efficient by some attempt at block anesthesia. Time and patience permits the use of less cocain and weaker solutions of adrenalin with better hemostatic effect.

If the effect of intranasal dressings or splints is for pressure, the cut areas may be protected by gutta percha or paraffin paper. Petrolatum does not favor coagulation. The effect may be more favorable with the addition of calomel or bismuth subnitrat. Some surgeons prefer narrow strips of gauze impregnated with bismuth powder. (The external application of cold following nose and throat operations may be mentioned in passing.)

At one period in my work, I ordered, routinely, hypodermic injection of ½ gr. of emetine following nasal operations. Dr. R. H. Rice, of Milwaukee, informed me that he prescribes 4 doses a day of potassium iodid, 2 grains, with bichlorid of mercury, gr. 1/30, for patients in whom the pathology suggests a predisposition to bleeding. For some years I gave calcium lactat tablets two days preceding tonsil operations. I have

also abandoned the local application of Monsel's solution, tannic acid, or tannic and gallic acid following tonsillectomies. Others may report medicinal remedies founded on empirical or laboratory data with experiences that favor their continued use.

Notwithstanding Feinberg's[1] paper on, "The Relation of Blood Coagulation to Postoperative Hemorrhage," as deducted from 500 tonsillectomies by various operators, the bleeding time of a person's blood when exposed to his cut tissues, and the coagulation time of his blood may be considered in the scientific study of medicinal remedies for the control of hemorrhage.

Grove and Vines[2] report the intramuscular (gluteal) injection of 1 grain of calcium chlorid in 100 minims of water of the greatest service in ordinary hemoptysis. Cushing[3] states, "That the small quantity of calcium absorbed from the alimentary canal has no obvious effects." Walters[4] reports reduction in coagulation time in cases of jaundice after the oral administration of 100 grains of calcium lactat daily for 5 days. He prepares patients with obstructive jaundice at the Mayo Clinic for operation by the daily intravenous injections of 5 cubic centimeters of a 10 per cent calcium chlorid solution for a three day period, and observes that in these patients the coagulation of the blood is greatly reduced and the toxicity diminished.

Mills[5] calls attention to tissue fibrinogen observing that, "Those tissues in which hemorrhage is most dangerous possess the richest stores of tissue fibrinogen—the brain, lungs, kidneys, and endothelium of the blood vessels." There exists one apparent contradiction to this view of the clotting process—the liver. This Mills attributes to the nature of the benzene soluble protein, phospholipin, which is a more active coagulent in the lung cells than in the liver.

Joseph B. Greene[6] has reported the activity of the clotting elements of tonsils made for him by Mills. I can subscribe in part to Dr. Greene's conclusions in that, "The application of the tonsil itself to the open vessels of the fossa controls bleeding more effectually than the usual method of sponging." I wish to resist the temptation of digressing to a consideration of the surgical technic. Suffice it to say, that time is an important element in the coagulation and bleeding process. Possibly the first two minutes of contact of the dis-

sected tonsil to the wound are of greatest value, but I have thought from my efforts that the following three or five minutes were also important. The tissue juices of value are not only the tonsil substance, and the tonsil capsule, but the patient's living tissues in the wound—the blood, the fascia, muscle and endothelium of the severed vessels. My observations would suggest that the mechanical extraction of the tissue juices by the artery forceps in oozing areas in the wound liberates the coagulating substance more effectively. Here, also, the few minutes of time of application are necessary for the various stages of clot formation. Starr[7], of Toronto, refers to the value of torsion. The tonsil wound is handicapped by the fluids of the mouth. Why then wash away more tissue juices by encouraging the patient after tonsillectomy to gargle with cold water?

As an acceptance of the action of tissue juices in coagulation, there are on the market several tissue extracts—thromboplastin (Squibb), hemolytic serum (Mulford), hemagulin (Lilly), obtained from calves brains, coaguline Ciba, coagulose, and hemostatic serum (Parke Davis & Co.), obtained from blood platelets, and also the plain horse serum. The laboratory reports given me following the use of both classes of these preparations showed shortened time of bleeding and coagulation. I do not feel that the series was sufficient to draw comparisons, but that clinical observations and laboratory figures indicate some value.

In cases of severe postoperative hemorrhage, my observations of the results of blood transfusion is that its effect is too well known to delay its use as a measure of last resort. The value of transfusion of whole blood is well stated by Peterson[8], "In acute hemorrhage, whether simple or pathologic, and in chronic posthemorrhage anemia, we have in blood transfusion the best and at times the only efficient remedy. As a tissue transplant to replace lost blood, as a hemostatic agent to check further bleeding, and as a stimulant to the hematopoetic system to manufacture new blood, the procedure is theoretically and practically sound." The intramuscular injection of untyped blood is an efficient remedy which usually is readily available.

Neuhof[9] carried out some experiments in 1916, consisting of the intravenous injections of solutions of sodium citrat into dogs under ether anesthesia. The following results are quoted:

"(1) The coagulation time is tremendously shortened a few minutes after the introduction of nontoxic doses of sodium citrat, and this shortened coagulation may be sustained for one or more days. (2) The bleeding time is likewise shortened, so that, after citrat injection, a large vessel can be incised with prompt coagulation about the wound. (3) Coincident with the shortened coagulation time, the color of the venous blood is altered to a light arterial tint. (4) There is no fixed toxic or lethal dose of sodium citrat per kilo of body weight, toxicity depending to a remarkable degree upon the rate of introduction of the citrat solution. (5) A toxic or lethal dose is characterized by a swing from the state of shortened coagulation to one of suspended coagulation. This latter phenomenon led to the sodium citrat method of blood transfusion, and it was this effect which overshadowed the ordinary pharmaologic action of sodium citrat."

Neuhof and Hirshfeld[9] report a series of 500 cases to whom sodium citrat has been administered, the last 200 being by the intramuscular route, and conclude that such administration results in prompt and pronounced shortening of coagulation and bleeding time, which is of two or three hours duration, with a gradual return to normal within twenty-four to forty-eight hours. They established the optimum dose as 30 c.c. of a 30 per cent solution, and the intramuscular route as the one of choice.

Following the work reported by Neuhof and Hirshfeld[9], in 1922, Dr. David Fisher and I have injected intramuscularly sodium citrat for the control (mostly prophylactic) of hemorrhage in over fifty patients in the Soldiers' Home Hospital in Milwaukee.

The coagulation time of the first twenty-five of this series is tabulated in the Report[10] of our work published in the August, 1924, number of the Annals of Surgery. Over twenty-five more injections have been carried out by my associate in the Soldiers Home Hospital, Dr. Eugene Dallwig, which, with injections given by me in private patients, runs the series over one hundred. No untoward results, other than temporary local tenderness, were observed following any of the injections. In one case there was considerable infiltration, but no fluctuation; we ascribed this to a hematoma.

BLOOD COAGULATION FOLLOWING THE INTRAMUSCULAR INJECTION OF SODIUM CITRAT*

Case Number	1	2	3	4	5
Coagulated time before injection	11 min.	9 min.	8 min.	6 min.	9 min.
10 minutes following injection..	9 min.	9 min.	7 min.	6 min.	8 min.
20 minutes following injection..	7 min.	7 min.	7 min.	4 min.	6 min.
30 minutes following injection..	4 min.	5 min.	4 min.	2 min.	4 min.
45 minutes following injection..	2 min.	2 min.	1½ min.	1 min	2½ min.
60 minutes following injection..	3 min.	2 min.	3 min.	3 min.	3 min.
6 hours following injection....	6 min.	3 min.	3 min.	3 min.	4 min.
12 hours following injection....	7 min.	5 min.	5 min.	4 min.	4 min.
18 hours following injection....	9 min.	6 min.	5 min.	4½ min.	5 min.
24 hours following injection....	9 min.	6 min.	7 min.	5 min.	7 min.
36 hours following injection....	10 min.	9 min.	8 min.	5 min.	9 min.
48 hours following injection....	10 min.	9 min.	8 min.	5 min.	9 min.

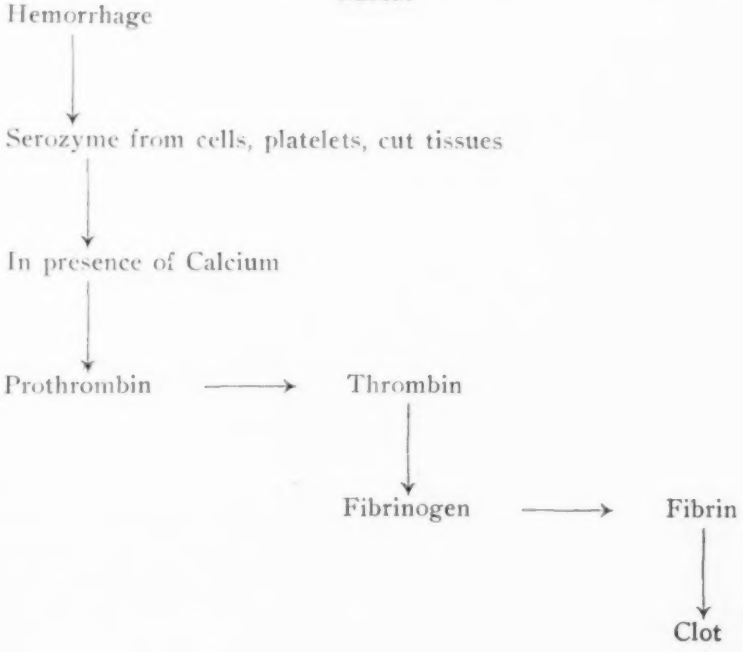
The chart of the first five cases of the series is typical of the drop in coagulation time of all the cases. Normal coagulation time of 3½ minutes is also shortened to coagulation time of 2 to 1½ minutes. In view of one septum patient who had some bleeding, and who was injected during the course of the operation, and the pleasing result of the other patients injected before operation, it is my opinion that sodium citrat is more effective when administrated before operation. If morphin or another narcotic is employed before operation, the citrat can be conveniently injected at that time. That it is of some value when used during an operation, is attested by its administration given during the course of a radical mastoid operation in a case of unusual, though not serious, oozing from the mastoid bone. In this instance, there was less sponging required than is usual, when completing the operation, some thirty minutes later, in the attic and middle ear.

The coagulation time of the first patient was eleven minutes on admission; ten minutes following the injection, the coagulation time dropped to nine minutes; twenty minutes after injection, the time was down to seven minutes; thirty

*This chart is a copy taken from Dr. Higgins' paper in the Annals of Surgery, August, 1924.

minutes after injection, the time was down to four minutes; forty-five minutes following injection, the lowest point was reached in this case, being two minutes. From then on there was a return to normal, but more gradual than the abrupt fall. One hour after injection, the coagulation time in this case was three minutes; six hours after injection, it was six minutes; twelve hours after injection, it was seven minutes; eighteen hours after injection, it was nine minutes; and so on until the coagulation time previous to the injection was reached. The field was drier following tonsillectomy in this case than the usual noncitrated case with normal coagulation time. This same patient was operated seven days later for deviated septum. His coagulation time then was nine minutes. If necessary the dose can be repeated in thirty-six hours, thereby controlling the coagulation time for a longer period. The majority of my cases were patients on whom nose and throat operations were performed, and the results were highly gratifying. The field of operation in tonsillectomy patients previously citrated was dry and remained so; sutures were rarely necessary.

OUTLINE SKETCH OF THE THEORY OF THE COAGULATION OF THE BLOOD.



Technic of the Intramuscular Injections.

The solution used is sodium citrat, C.P., 30 per cent, sterilized by boiling. The dose given is 30 c.c., and the site of injection is the buttocks. The skin over each buttock is cleansed with alcohol, dried, and tinctur of iodin applied over a broad area. With a three inch needle, 3 c.c. of a 1 per cent novocain solution is injected into each buttock. Three minutes are allowed to elapse, and then 15 c.c. of the citrat solution are injected into each buttock, directly in the same area as in the injection of novocain. The novocain tends to diminish to a great degree the pain, which is temporary only. Where time is an important factor, or where the condition of the patient is such that he cannot be moved, the entire 30 c.c. may be given in one buttock. I have injected 30 c.c. in one buttock on five different occasions; in the case of mastoiditis, and in four tonsillectomies following peritonsillar abscess. Tenderness was noticed in a few instances; but this was relieved even in the sitting position by a soft pillow or rubber ring. I also used citrat intramuscularly in consultation in a severe case of postoperative hemorrhage, in which horse serum and similar agents were administered before the citrat injection.

Intravenous Injections.

Neuhof and Hirschfeld[9] indicated that toxicity depends upon the rate of introduction of the citrat solution. The intramuscular route seemed to be the one of choice for slow absorption and optimum dosage. From a personal use of sodium citrat intramuscularly in 100 cases, I feel that its application in this manner is safe and efficient. The recent work of Rosenthal and Baehr[11] indicates that sodium citrat may be safely administered intravenously. They state that if properly administered, from 3 to 6 grams can be safely given to an adult of average size. The citrat should be well diluted, and the procedure should take at least fifteen minutes continuously, not intermittently, during that period, to avoid too great a concentration in the heart's blood at any one time. They report that they have repeatedly employed this means of arresting secondary hemorrhages in a variety of medical conditions, such as gastric ulcer, pulmonary tuberculosis, cerebral hemorrhage, typhoid fever, and after tonsillectomy. From 3 to 6 c. c. of a 30 per cent solution of sodium citrat (3 to 6 gm.) has usually been administered, very slowly, the

intravenous injection taking from ten to fifteen minutes for completion. In concluding their paper, Rosenthal and Baehr advise, "For absolute safety the administration of sodium citrat in from 200 to 500 c.c. of blood in the form of the citrat transfusion of Lewisohn."

Nature of the Reactions.

Rosenthal and Baehr's extensive experiments show that:

1. Sodium citrat does not destroy the blood platelets in the test tube, but rather it preserves them, so that they may be more readily counted.

2. Within a few minutes following the intravenous injection of sodium citrat, the blood platelets diminish in number; the maximum decrease is usually within ten to fifteen minutes, with return to normal number in one-half to one hour.

3. Free thromboplastin substance (cytozyme, serozyme) appears in the blood as the coagulation time is shortened.

4. No demonstrable changes in the blood were obtainable in regard to the other factors in coagulation, calcium, fibrinogen, or antithrombin.

5. Shortened coagulation time and increase in the thromboplastic agent follows the reduction in number of blood platelets.

6. Coagulation time is not shortened by the intravenous injection of sodium citrat in animals (ducks) which are devoid of blood platelets; the coagulation time was in one case greatly prolonged.

7. Coagulation time is *prolonged* following the introduction of sodium citrat in human beings in the presence of diseases deficient in blood platelets, numerically or qualitatively, e.g., purpura hemorrhagica and congenital hemophilia, or pernicious anemia, or Bantés disease. They conclude that the action of sodium citrat is upon the blood platelets not by direct destruction, but that the blood platelets are damaged, and then removed from the circulation by some organ, probably the spleen, there destroyed, and their thromboplastic substance liberated into the blood stream.

Renaud and Juge[12], in Paris, report the observation of 14 cases, in which the injection of solutions of sodium citrat has been followed by an immediate and abrupt cessation of profuse hemorrhage, which had resisted all other therapeutic measures. After injecting 10 to 25 c.c. of a 30 per cent solu-

tion of sodium citrat, freshly prepared, they have witnessed in the great majority of cases (14 out of 17) an almost immediate cessation of hemorrhage, such as occurs in cancer and tuberculosis. They observed a reaction following the intravenous injection of sodium citrat—the symptoms were anxiety, malaise, increased pulse rate with a fall in pulse tension and nausea. Dr. Dallwig injected intravenously in one patient 200 c.c. of a 3 per cent solution. The patient complained of fleeting symptoms during the injection, tingling sensations in the extremities, nausea, and slight faintness. Possibly the symptoms of a reaction may be avoided by prolonging the time of administering the injection.

Conclusions.

1. Many medicinal remedies have value in the control of hemorrhage.

2. The value of sodium citrat is clinically and theoretically proven.

3. More general use of sodium citrat will establish proficiency and refinements in the administration.

BIBLIOGRAPHY.

1. Feinberg, Samuel M. The Relation of Blood Coagulation Time to Postoperative Hemorrhage. Surgery, Gynecology and Obstetrics, March, 1924, vol. XXXVIII, p. 363.
2. Grove, W. R., and Vine, H. W. C. The Control of Hemorrhage by Intramuscular Injection of Calcium Chlorid. British Medical Journal, July 9, 1921, p. 40.
3. Cushing, A. R. The Action of Drugs in Health and Disease. Philadelphia. Lea and Febiger, 1913.
4. Walters, Waltman. Preoperative Preparation of Patients with Obstructive Jaundice. Surgery, Gynecology and Obstetrics, December, 1921, p. 651.
5. Mills, C. A. Blood Clotting and the Control of Hemorrhage. Medical Record, January 28, 1922, p. 149.
6. Greene, Joseph B. Further Experience in the Use of Tissue Juices in Tonsillectomy. Annals of Otology, Rhinology, and Laryngology, March, 1924, vol. XXXIII, p. 228.
7. Starr, F. N. G. Postoperative Hemorrhage. Surgery, Gynecology, and Obstetrics, March, 1924, vol. XXXVIII, p. 356.
8. Peterson, Edward W. The Value and Limitations of Blood Transfusion. Annals of Surgery, 1923, vol. LXXVII, p. 364.
9. Neuhoff, Harold, and Hirschfeld, Samuel. Annals of Surgery, July, 1922, vol. XXV, p. 1.
10. Higgins, Samuel G., and Fisher, David. Effects of the Intramuscular Injection of Sodium Citrat Upon Bleeding. Annals of Surgery, August, 1924, p. 268.
11. Rosenthal, Nathan, and Baehr, George. Paradoxical Shortening of the Coagulation Time of the Blood after Intravenous Administration of Sodium Citrat. Archives of Internal Medicine, May, 1924, vol. XXXIII, p. 535.
12. Renaud, Maurice, and Juge. On the Hemostatic Action of Citrat of Sodium. Comptes Rendus de la Société de Biologie, Paris, February 22, 1924, vol. XC, p. 425.

DISCUSSION.

DR. J. J. KING, New York City: As a rule, there is more bleeding in operations done in a state of acute inflammation than when the operation is done in a state of chronic inflammation. For instance, in a mastoid operation, and in many of the acute operations which we have designated hemorrhagic, the bleeding is very profuse, and if Dr. Higgins has brought forth a remedy which will lessen the bleeding in these operations, he has rendered us a distinct service. In my experience, I have had to depend more upon good judgment in operations, and the carrying out of good surgical technic to control hemorrhage or to prevent it, than upon any medical remedies I have used. In fact, the more I have thought about this subject, the more I have felt that so far as my experience is concerned, there is no medical remedy upon which we can depend to control hemorrhage except such things as adrenalin, hot salines in the wound, and mechanical means. I cannot recall any great benefit received from injecting drugs into the body, or from administering them by mouth, in controlling hemorrhage. So I have concluded that I must depend more on good surgical technic in carrying out the operation than upon medical remedies.

I do hope, however, that the injection of sodium citrat will prove to be the royal remedy that Dr. Higgins feels it will. If it does, it will be a great benefit in our work. Being chemical, it has the advantage of not being toxic like serums and various other preparations, which may cause anaphylaxis.

I think there is not much danger of such a report creating too much confidence among surgeons, because, with experience, they know we cannot depend upon medical remedies, but must depend upon good surgical technic. I am afraid that if a layman read this paper, he might think he could cut a blood vessel and this would control the hemorrhage, but it will not do that.

DR. SECORD H. LARGE, Cleveland: I have been trying to think how many cases of hemophilia we have seen in our practice. I cannot recall one case. Last year, we reported 3,000 tonsillectomies performed in our dispensary. Of these, there were only four cases in which the blood clotting time was over ten minutes. These cases were given calcium chlorid or calcium lactat by mouth, and they had no hemorrhage either during or after the operation. Certainly, these cases were not hemophiliacs, inasmuch as these drugs have been proved to be practically of no use when given by mouth.

In performing operations on the accessory sinuses and the mastoid, we find that hemorrhage is better controlled by packing in gauze which has been dipped in hydrogen peroxid than by the use of adrenalin. I have had no experience with sodium citrat, so I am unable to pass judgment on it, but as Dr. Higgins describes it, it certainly should take the place of serums.

DR. GEORGE F. KEIPER, Lafayette, Ind.: Some of the worst hemorrhages we see are due to injuries common to patients in our shops, especially where they are struck across the nose by a heavy piece of metal, and ofttimes these are very hard to control. We find it is best in such emergencies to take time by the forelock, by securing a proper donor and making a transfusion of blood, using the paraffin tube.

We have all been using chlorid of calcium or lactat of calcium by mouth, but we certainly have a valuable suggestion here. Let us not forget the fact, in the removal of tonsils, that someone has told us we should put the tonsil immediately back into the fossa before removing it from the mouth, and that that would go a great way toward controlling hemorrhage. The use of organic hemostatics is not without danger, and we may take a cue from workers in other lines; for instance, DeLee, in the Journal of the American Medical Association of May 10th, tells of a case of postpartum hemorrhage he had which was controlled by packing. The packing was removed and the hemorrhage returned. The womb was packed again with hemoplastin on gauze, and the woman was dead in six minutes. She was an asthmatic. In the Journal of August 30th, 1924, Dr. Hektoen reports on the following coagulents: Coagulen-Ciba, Fibrogen-Merrell, Hemagulen-Lilly, Kephalin-Armour, Hemostatic Serum Lapenta (Hemoplastin)—P. D. & Co., Thromplastin-Lederle, Thromboplastin-Squibb, Thromboplastin-Solution-Armour, Precipitated Horse Serum (Coagulose)—P. D. & Co. It was found that all of the specimens contained animal protein; in most products, horse or beef proteins were present. In most cases, the labels and descriptive literature did not state the precise nature of the coagulant. His suggestion is, that all such preparations should be labeled properly, but it is up to us to make inquiry into the patient's history to find out whether any peculiar sensitiveness exists, before the administration of these remedies.

Dr. Sidney Israel, Houston, Texas: I think there is a tendency sometimes to forget the essentials of surgery, and to expect to operate without seeing a drop of blood. If we recall our earlier hospital days, during the time we served as internes or on the House Staff, it will be remembered, that in all general operative work bleeding took place, but was promptly controlled either by ligature or suture. In our practice, we have carried out investigations for about six years on the relative value of coagulation and bleeding time, especially in relation to the tonsil operation. We have also investigated the various methods of testing the coagulation time, in order to arrive at the one that was most reliable or accurate with regard to the clinical findings at the time of operation.

Hemophilia is rare in our experience, and one of the most important points in the suspicion of hemophilia or bleeder is the history, from the patient, which can usually be ascertained prior to any operative procedure. Most of the coagulation tests are fairly accurate and satisfactory, provided they are carried out by a capable observer with a standardized technic.

We have seen instances where the coagulation and bleeding time was normal, but in spite of these facts, an excessive amount of bleeding took place at the time of operation. This we can explain as being due to the arrangement of the blood supply in these instances, regardless of the fact that the clotting and bleeding time were well within the normal time. It is cases such as these, that convince us that any drugs applied to the tonsil fossa with the idea of controlling hemorrhage or profuse bleeding is a great mistake, and only takes us further away

from sound surgical principles, always to be kept uppermost in the mind of the laryngologic surgeon.

If scrupulous care in observation and examination is carried out prior to operation, and if the operator possesses the proper skill, there should be little risk insofar as alarming tonsil hemorrhage is concerned.

The use of sodium citrat, as outlined by Dr. Higgins, is a most valuable contribution, and should be carried out when indicated, though its use in cases following tonsillectomy would be relatively few.

Dr. Samuel G. Higgins, Milwaukee (closing): I am pleased to see that the discussers are in accord with what I have written: I hope the paper has emphasized our surgical responsibility, and yet has given us some hope of medical control when the inevitable hemorrhage does occur.

Acute inflammation does have an effect upon blood platelets and upon the structure of the blood, which, by further reading, we learn contributes to the cause of delayed coagulation. I feel that surgical and medical judgment must be used in operating and in determining the choice and time for operation.

TREATMENT OF DEFORMITIES OF THE NOSE DUE TO ACCIDENTAL AND OPERATIVE TRAUMATISM.

WILLIAM WESLEY CARTER, A.M., M.D., F.A.C.S.

NEW YORK CITY.

Basic knowledge upon any subject connected with either medicine or surgery is dependent upon a clear conception of the anatomy of the parts involved, and this knowledge is greatly enhanced, and its practical application facilitated, by a fair understanding of the embryology and developmental history of the organ under consideration. Especially is this so in the case of injuries occurring before the age of mature development.

Papers on plastic surgery, as a rule, deal only with cosmetic results, and are content with a diagrammatic demonstration of procedures and before and after photographic illustrations. Now this subject is of sufficient importance to deserve more scientific treatment, and it seems to me that it is a mistake to ignore basic factors which contribute so largely to determining the character of the deformity following an injury to the nose. It is my purpose here, however, to refer only briefly to those factors that are of practical clinical importance, and that will explain satisfactorily the type of deformity following an injury to the nose, and suggest the principles that should be observed in correcting it.

It must be kept in mind, that the rigid and semirigid framework which supports the soft tissues of the nose is an arch, both from an architectural and mechanical point of view; that is, it is a curved structure capable of preserving its integrity, though supported at its extremities only. The septum strengthens, but is not indispensable as a prop to the arch. Its upper edge, however, where it fits in between the two lateral cartilages, forms the keystone of the arch.

A brief review of the anatomy and development of the nose will enable us to appreciate more fully the changes which occur in the relations, which the several parts which make

up the nasal arch bear to each other, when fracture or dislocation has occurred.

The walls of the nose proper are formed above by the nasal bones, the nasal spine of the frontal bone and the nasal processes of the superior maxillae, and below by the paired lateral and greater alar cartilages. Accessory elements are the accessory alar cartilages and the sesamoid cartilages. Above, the skin is loosely adherent, but below it is closely attached over the alae and lobules. The unpaired septal cartilage and the vomeronasal cartilages of Jacobson assist both directly and indirectly in supporting the external nose. The osseous portion of the septum is formed by the perpendicular plate of the ethmoid, the vomer, the nasal spine of the frontal, the rostrum of the sphenoid and the crests of the nasal, maxillary and palate bones.

The blood supply comes from the nasal, angular, infraorbital, lateral nasal and superior coronary arteries. The free anastomosis of these numerous vessels, coming as they do from different trunks, gives such an abundant supply of blood that portions of the nose may be almost completely severed, and yet they will retain their vitality. Instances have been recorded where a large portion of the nose, having been completely severed from the body, has been replaced, and perfect union and recovery from the injury have followed. It is said that in India, where punishment for certain crimes at one time consisted in cutting off the nose, the law had to be amended so as to include the destruction of the severed organ, as it was found that the victims were successfully grafting on their noses, and thus escaping to a large extent the intended punishment. This remarkable blood supply makes this field an ideal one for plastic surgery; infection is resisted, flaps do not lose their vitality, union is rapid and secure, and there is a minimum of scarring.

The nerve supply of the nose proper is derived from the nasal, infratrochlear and infraorbital nerves. The muscles are supplied by the facial.

I have been interested for a number of years in the dynamics of nasal development, and have obtained much valuable information of practical clinical value by studying the forces which change the flattened nose of the infant into the more shapely and prominent organ of the adult. My studies lead me to believe that this is effected chiefly by the vomer, acting

upon the other structures of the septum, which raises the bridge of the infantile nose like a tent pole, the sloping walls following and forming a pyramidal structure. The strategic position of the vomer is impressive, being placed like a vertical wedge between the unyielding base of the skull and the palatal processes of the superior maxillae and palate bones. Its anterior edge is an inclined plane, sloping downward and forward, and is parallel with the prospective contour of the developed nasal bridge; into this edge are fitted the vertical plate of the ethmoid and the septal cartilages. Now, just as we may learn the character, strength, direction, etc. of forces by studying their effect upon inanimate matter, so may we learn the influence which the septum has upon the development of the nose by observing the effect which strain has upon this structure in the performance of its normal function, that of raising the bridge of the nose, and conversely by

Fig. 1. Case 1. Traumatic deformity due to injury in early life. Note flattened appearance due to absence of septal influence during developmental age. Before correction.

observing the effect upon the development of the nose, when from any cause the performance of this function of the septum is interfered with in early life. While it is very true that developmental processes go on hand in hand, it is also true that in some portions of the body there is normally an unequal growth of related parts, which results in moulding and developing the infantile type into the adult type. At birth, the infant's nose is flat and stubby, and the nasal fossae are as wide as they are high. The posterior nares are relatively small, the vertical diameter being 5 to 7 mm., and the transverse diameter being 7 to 8 mm. By the sixth month of extrauterine life, these diameters have doubled, but the choanae are still more or less circular. At puberty, the bridge of the nose has been raised and the organ has acquired its adult proportions. The vertical diameters of both the nasal cavities and the choanae are longer than the transverse. The

Fig. 2. Case 1. (Same as preceding.) Corrected by bone and cartilage transplantation.

changes are due to the unequal growth of the related parts. It is my belief, based upon clinical and osteologic study, that the septum has more to do with the shape and contour of the nose and the position of the hard palate than any other factor.

The development of the vomer throws important light upon the formation of the nose, especially from a cosmetic point of view. The vomer is developed, not from the primitive cartilaginous septum, but from two centers of ossification which appear about the eighth week of fetal life, one on either side in the membrane which covers this cartilage. The vomer develops therefore as two plates, which enclose between them the primitive cartilage. These two plates begin to unite posteriorly, and their coalescence gradually extends forward, the primitive cartilages being absorbed and to a certain extent pushed forward, thus assisting in raising the nasal bridge. It is significant that the two plates of the vomer are not united until the fifteenth year, when the nose has assumed its adult

Fig. 3. Case 1. Profile of same patient before operation.

proportions. When union between the two plates is not complete, we have the bilaminar vomer; in such cases, the bridge of the nose is very low. Where there is no union between the plates, there is a sulcus between the two nasal cavities, producing the deformity known as wolf nose. If this explanation of the development of the nose be accepted, the frequency of deviated septa, 75% in the white races, is easily accounted for, as the strain upon the septum in causing the pronounced elevation of the nasal bridge, characteristic of these races, is very great, and deviation from the vertical following some slight traumatism is very apt to occur, especially during the developmental period. At the same time, the deviated septum offering little or no resistance to the hard palate, the latter becomes high arched and narrow, and the upper jaw deformed. Just as in any mechanical structure, an imbalance of forces produces distortion.

Fig 4. Case 1. Profile after correction.

Fig. 5. Case 2. Compound comminuted fracture two years after injury.

Fig. 6. Case 2. (Same as preceding.) Corrected by bone and cartilage transplantation.

The extent of these developmental forces, their direction and the results accomplished when acting normally upon the nose in its developmental stage, must be kept constantly in mind when treating nasal deformities, whether they result from traumatism, disease or developmental irregularities. In traumatic nasal deformities, the lower ends of the nasal bones and the septum are usually fractured and dislocated; if dislocated upwards over the nasal spine, they are extremely difficult to reduce without an open operation. The nasal processes of the superior maxillae may be involved together with the lacrimal bones. Here, stricture of the lacrimal duct is almost certain to follow, sometimes despite the most judicious treatment. The upper ends of the nasal bones are seldom fractured; traumatism at this point is more apt to cause fracture of the cribriform plate.

Deformities following the submucous operation are becoming so frequent that this may be well regarded as one of the causes of saddle back deformity. A fair number of these lamentable accidents are avoidable, because they are due to the attempt on the part of men to do this operation, who are unfamiliar with the proper technic, who are untrained clinically, or who naturally do not possess the special quality of manual dexterity which this artistic little operation seems to demand. Anyway, men are doing this operation who ought not to be doing it. I say this, because of the increasing number of deformities following this operation that are coming to me for correction. It is said that one of our well known American surgeons, while abroad, attended the clinic of a German rhinologist distinguished for his voluminous writings. This doctor was doing a submucous operation, when he displaced the upper edge of the septum from its position between the lateral cartilages, where it performs the function of the keystone of the nasal arch. He recognized the mistake at once, and, anticipating what would be the result, removed a sliver of bone from the tibia and transplanted it into the nose, naively remarking that he always prepared the tibia beforehand in anticipation of just such an emergency.

It is needless for me to say that men of this type should not be doing the submucous operation. This procedure seems to demand a special kind of dexterity, for it is a well known fact that some operators, highly skilled in other fields of surgery, cannot do a satisfactory submucous operation to save

their lives. If properly performed upon an adult, aside from the unavoidable risks which attend every surgical procedure, great or small, the submucous operation does not endanger the nose, from either a functional or cosmetic point of view, nor is its structure weakened. In view of the importance of this subject, you may pardon me for emphasizing some of the well known precautions that should be taken when performing this operation: No patient under fifteen years of age should have any considerable amount of his septum removed. If it is absolutely necessary to operate, take out only a narrow strip. Take every precaution to avoid hematoma and sepsis. Great care must be exercised in removing the septum after the mucous membrane has been elevated; only those portions which are causing obstruction should be removed and a liberal ridge pole of cartilage should be left. Do not use a tugging instrument, as this is apt to dislodge the upper edge of the septum. For this reason, mainly, I have never advocated the use of the swivel knife. I have always used the punch forceps I devised for this purpose. With this instrument, there is no tugging, and the exact amount and location of the tissue to be removed can be acurately determined. (Fig. 13.)

Abscess and hematoma of the submucous sac are to be carefully guarded against, as they are almost certain to be followed by a depressed deformity. In my experience, the occurrence of hematoma has been greatly limited by the use of my gold wire splints instead of packing. Not only do they add greatly to the comfort of the patient, by enabling him to breathe through his nose after the operation, but they exert continuous pressure on the flaps and prevent hemorrhage. In those cases where the upper edge of the septum has been accidentally displaced or removed while doing the submucous operation, a transplant from the rib should, if possible, be introduced at once.

In abscess of the septum, no attempt should be made to correct the deformity until complete healing has occurred, and we are quite sure that no infection remains, and that the granulation tissue which lined the walls of the abscess cavity has become organized, for bone transplants will not tolerate granulation tissue.

The technic of my operation has been fully described, and I will not discuss it in detail on this occasion; suffice it to say, that the bone and cartilage transplants are taken from the

rib and are introduced through a small slit in the vestibule of the nose, after the tissues over the dorsum have been elevated. As a rule, I use a combination of bone and cartilage, and not only shape the transplant to suit the deformity, but endeavor to replace the lost bone and cartilage with similar tissues, so that the result will be truly a physiologic reconstruction of the nose. In a general way, I may say that I use bone where active support is needed, since this tissue not only retains its vitality, but grows, its growth being regulated by the physiologic demands of the part; while cartilage, which remains just as it was when introduced, is used chiefly as a filler for the correction of irregularities in the contour. Neither bone nor cartilage, when properly transplanted, is absorbed, as I have been able to demonstrate on many occasions by X-ray

Fig. 7. Case 3. Full face. Entire tip of nose destroyed (bitten off)

plates of numerous cases operated upon many years ago. By this method, the results secured are permanent, and the patient is not subject to the menace of a foreign body in the tissues. The intranasal operation, while it demands greater skill on the part of the operator and more perfect technic, is far preferable to any external incision, as no noticeable scar results. I have long since abandoned the external incision.

In the case of fresh fractures with dislocation, I wish to emphasize here the importance of prompt adjustment and immobilization; the keynote of efficient service is the prevention of deformity. In many of these cases, I am still using my bridge splint with gratifying results. In the old traumatic deformities, cases where the fractures were never properly set,

Fig. 8. Case 3. Same case (profile). This deformity was corrected by a flap from the forehead. (Photo after operation could not be secured for publication. Result was perfect.)

Fig. 9. Case 4. Full face. Child, 12 years of age. Deformity due to absence of developmental influence of septum caused by an injury when she was two years of age.

Fig. 10. Case 4. Full face. After correction by intranasal shifting of tissue and transplantation of bone and cartilage. Two years after operation.

Fig. 9. Case 4. Full face. Child, 12 years of age. Deformity due to absence of developmental influence of septum caused by an injury when she was two years of age.

Fig. 10. Case 4. Full face. After correction by intranasal shifting of tissue and transplantation of bone and cartilage. Two years after operation.

Fig. 11. Case 4. Profile before operation.

Fig. 12. Case 4. Profile after operation.

and where there is always to be found an abundance of scar tissue, the bridge splint may be used after thoroughly mobilizing the entire nose, but the results are not always satisfactory, as the contraction of the scar tissue and the elasticity of the skin tend to reproduce the original deformity when the bridge splint is removed, and bone transplantation is the final recourse.

In recent injuries, where there is extensive destruction of the soft parts, great care should be taken to save all remaining tissue, for the extraordinary cellular vitality exhibited in this area will enable us to use contused and even macerated soft parts which in any other part of the body would be hopelessly lost. Where there is moderate destruction of the nose, I employ shifting of tissue, Thiersch and whole skin grafting. In the latter, skin is often taken from the back of the neck. When the destruction is more complete, I use a flap from the forehead. First I make a pattern of the size and shape desired out of adhesive plaster; this is placed on the forehead, the pedicle corresponding to the source of the best blood supply, the inner angle of the orbit. I neither use nor recommend the socalled tubular flap, as the making of the flap into a tube tends to constrict the blood supply. If the raw surfaces of the flap are thoroughly covered with vaseline, it is amply protected from the air, and at the same time the integrity of the exposed blood vessels is preserved by the vaseline. The use of the adhesive plaster pattern, as originally suggested by me, greatly facilitates accuracy in cutting the flap. Pedicle connections should not be severed under two weeks. The practical application of my methods can best be illustrated by the figures thrown upon the screen.

In conclusion I will say that it is important that a surgeon dealing with deformities of this organ should have not only an intimate knowledge of the osseous and cartilaginous framework of the nose, but also of the nasal muscles, nerves and blood supply. He should also have that artistic endowment which will enable him to recognize the proper relations which the nose should bear to its facial environment, and he should have the manual dexterity to enable him to establish those relations. These requirements are, to a large extent, individual endowments, and they place the practice of plastic surgery of the nose and face upon a scientific basis and a plane beyond the reach of many, whose only claim to recognition in this field is a trip to Europe and a fanciful experience with foreign operators, which they so vividly describe in illuminated type.

Plastic surgery was for many years exclusively in the hands of the charlatan, and even now is tainted by his polluting touch, and I appeal to this body of scientific men to advocate its importance and to defend its dignified position, and thus increase its field of usefulness.

Fig. 13. Submucous instruments for removal of the nasal septum without weakening the nasal arch.

DISCUSSION.

Dr. J. J. Sullivan, Jr., Scranton, Pa.: We can always expect something worth while when we hear from Dr. Carter. Anyone who does an 80 per cent correction in submucous resection is bound to have a slight drop in a small percentage of cases. I am speaking now of men skilled in their specialty.

I agree with Dr. Carter as to the age limit in the young. I think he was one of the first to describe the socalled arch or keystone of the nasal septum. If you are careless enough to remove it, or rough enough to displace it, you will have a drop.

But, how about some of our cases where it is left; where a large amount of cartilage remains, and still we get a drop? That one part bothered me very much for a long time, until we began to check up and found the trouble. Nearly all these cases, if you will notice closely, have a slight drop, or when nose is twisted to one side have a slight drop.

Example: A young man, deflected septum, examination of nose disclosed drop. Over ¼-inch of septal cartilage allowed to remain. Arch untouched, no drop until nasal spine was removed, and then a drop. We do not allow any of these cases to leave the office in this condition. So we inserted a piece of cartilage or bone removed during submucous resection, with very satisfactory result.

Dr. John W. Fairing, Greensburg, Pennsylvania: Some twelve years ago, while working with Dr. Carter at the Manhattan Eye, Ear and Throat Hospital, we had a case that came from Rochester, New York. This woman had absolutely no nose, and came to the hospital, on Dr. Carter's service, wearing a pair of spectacles to which was at-

tached an artificial nose. Dr. Carter did a four stage operation in repairing the damage. He buried a piece of split rib in the right arm and allowed that to heal. About two weeks later, he dissected the implant partially from the overlaying tissue and attached that immediately beneath the glabella, with the arm up in front of the face. Later, further dissection was done, and the implant fixed laterally on either side, and then a piece of rib bone fashioned to hold the nose out at the tip. This is the point I wish to make. Immediately after the flap was severed from the arm, which was twelve days after the original operation, it became cold and pale, and it was evident it would be lost unless some radical measures were adopted at once that would insure adequate circulation. Dr. Carter suggested that a leech be applied to the distal end of the flap. This we succeeded in doing, and much to our gratification, the flap immediately assumed a healthy color as the leech began to fill up. There was no further trouble, and the case progressed to a successful termination.

Dr. Samuel Salinger, Chicago: It is not often that one has an opportunity to find out what becomes of cartilage and bony transplants—I mean histologically. I recently put a rib transplant into a nose for the correction of a deformity due to an abscess of the septum. This transplant took, and the patient had a fairly good cosmetic result. About four months later, he died of an intercurrent trouble. At autopsy, we took out the part of the nose in which we had transplanted the cartilage, and I have a piece of it here. It shows two strips of cartilage, cut in section, in the region of the septum, still embedded in fibrous tissue which attaches the transplant to the lateral cartilages of the nose. I am having it stained and hope to get some information about how this cartilage is nourished.

One of the points Dr. Carter brought out, which is worthy of emphasis, is the necessity for immediate replacement of the fragments after fracture of the nose, and their proper retention.

I have had a large experience in fractures of the nose at the Cook County Hospital, and was impelled some time ago to devise a splint for this purpose. This acts on the same principle as Dr. Carter's, in that we get intranasal support of the bridge, and it also has the added factor of fixation of the splint to the forehead. It also applies pressure on the outside of the nose when required, without disturbing the intranasal portion.

Dr. J. M. Banister, Omaha, Nebraska: To the importance of this subject we can well subscribe, because those of us who have dealt with these cases find that so many are monomaniacs on the subject of their deformity. They grieve over it and are afraid to go into society. Anything that will serve to place such persons in proper mental relation to their environment is a matter of great benefit to humanity. About seven or eight years ago, I had my attention directed to Dr. Carter's methods, and I have been using them ever since.

A few points in regard to the technic that the Doctor did not touch upon. So many people have asked me about removing the rib. The great trouble with specialists beginning this work is, that they have a tendency to take out too small a piece. I think that no less than two inches should be removed, and it would not hurt to take out two and

a half inches. If you take out less than two inches of rib and make a transplant, it will be found to be too short, as a rule. Anyone who has done general surgery need not fear the consequences of rib resection. The principle is to cut directly through the periosteum for the length of the incision in the soft tissue, and shell the rib out of the periosteum. I make no attempt to preserve the periosteum, but remove the rib entirely. The rib is resected, divided into anterior and posterior halves, and the cancellous tissue curetted out. One then has a piece of bone that can be used in any way desired. These grafts will hold. I have just had the good fortune to see an individual upon whom I operated five years ago. I could see the whole outline of the bone insert transplanted five years before. He is quite an athletic fellow, and told me when last seen, that he had been struck many times over the nose in boxing, but the graft was solid yet.

We are all indebted to Dr. Carter for explaining why it is not wise to do a submucous resection of the septum in the case of the young. He has shown us the importance of preserving the osteogenic centers in such individuals. One can do a submucous operation for lateral deformity, due to malformation of the cartilaginous septum, without removing any of the tissue, and I have done it for a number of years This operation can be performed on children with absolute impunity because such an operation does not interfere with the osteogenic centers.

Dr. E. H. Porter, Tiffin, Ohio: Within the last two days, I saw the patient with the tip of the nose broken off, whose picture Dr. Carter showed. He was absolutely in perfect shape. He tried to mix white mule and a high powered automobile, and something happened—and Dr. Carter did the rest.

Dr. W. W. Carter, New York City (closing): I think a very important point is brought out by Dr. Sullivan, and that is, that there is in some cases a slight drop of the nasal bridge after a septum operation. This should not be. It is because either too much of the septum has been removed, or the upper edge of the septum has been displaced.

The operation of Dr. Banister is interesting, and I think should be a general check up on the rhinologist, because it is important to preserve as much of the tissue of the septum as possible, which Dr. Banister does. I have had great success in doing his operation.

The idea of introducing a piece of septal cartilage into the dorsum of the nose is to fill out a depression. This piece of cartilage should be put in very accurately, otherwise it will cause an irregularity. I do not know whether it remains permanently or not. I have an idea that the cartilage and bone of the septum are absorbed, because they are of membranous origin and not from osteogenetic centers.

I am very much interested in the specimen shown by Dr. Salinger, and will be glad to hear from him later when he has made a microscopic report.

Dr. Banister is right in saying that we should take a sufficiently large section of rib. I am glad, too, to note that he is one of the men who emphasize the fact, which has been established beyond all peradventure of doubt, that bone transplanted properly into the nose is not absorbed. This is the experience of all accurate observers who have watched their cases over a number of years.

THE GRADENIGO SYNDROME WITH THE REPORT OF THREE CASES.

WILLIAM HARDIN SEARS, M. D. F. A. C. S.,

HUNTINGDON, PA.

Isolated paralysis of the external rectus complicating acute suppuration in the temporal bone had been reported in the literature a number of times prior to Gradenigo's[1] presentation of his syndrome, which added to these head pains of an unusually severe and persistent type.

The original paper (1904) incorporated case reports of several patients of his own and others collected from the literature. From these, the deduction was made that this triad—acute suppurative otitis media, severe temporoparietal pains and paresis or paralysis of the abducens of the same side—constituted the symptom complex of a definite clinical entity.

In a further publication, three years later, the same author tabulated the records of all cases appearing in the literature to that date, 57 in number; to these he added a discussion of 8 cases in considerable detail, and a general consideration of the various points involved.

The writer ventures to insert, for the benefit of those who have not read the original, a rather literal translation of Gradenigo's[2] interpretation of the symptoms making up the so-called triad, and, also, the conclusions drawn from his study of the cases mentioned.

"We are dealing, therefore, with 57 cases, of which 24 belong to the first, 29 to the second, and only four to the third category."

In seven cases C. O. M. S. was present; the acute inflammations are therefore in the majority (87%).

The mortality is also very low, for there were only four deaths in 57 cases. In 92% recovery occurs after operation in the region of the mastoid process, or in the cranial cavity, as well as without operation. It can, therefore, be maintained that the disease usually occurs in connection with an

acute otitis, and that in the majority of cases the prognosis is favorable.

In 55 cases in which the sex is indicated, there are 37 men (67%) and only 18 women.

In 49 cases in which the side of the body on which the infection occurred was taken into account, it was 28 times on the right and 21 times on the left. The infection, therefore, occurs in a slight majority of cases on the right side (57%).

In reference to age, the following results are given in 50 cases:

Age.		Age.	
1 to 10	12	41 to 50	5
11 to 20	10	51 to 60	0
21 to 30	12	61 to 70	1
31 to 40	9	Above 60 years	1

The affection, therefore, occurs in the majority of cases in the first decennium, especially the first and third, which fact coincides in the main with statistics of ear diseases.

Let us now examine in detail the several symptoms:

Acute Purulent Otitis. Usually there are symptoms of retention of pus in the tympanic cavity, because of a complete lack, or insufficiency, of perforation of the tympanic membrane. In 32 cases, no spontaneous perforation occurred or was very late, or paracentesis was performed, but likewise quite late. The perforation was for the most part too small and insufficient for drainage, so that often an artificial extension of the same was necessary; aside from that, the opening of the tympanic membrane often showed an inclination to close, so that the paracentesis had to be repeated, in one case even five times during the illness.

A noteworthy circumstance to which I would like to call attention, so that others in the future may take notice of the same, is the relatively frequent site of the perforation in the anterior half of the drum, for this, as is well known, occurs rarely in acute inflammation.

In 14 cases in which the site of the perforation was given by the author with exactness, it was in eight cases in the anterior part of the membrane, five times in the anterior inferior, one time in anterior superior, and two times in the anterior half. The paracentesis or the extension of the perforation is usually followed immediately by relief of the other important symptoms; that is to say, pain and paralysis. A case of Picks

and another of Cozzolino are highly illuminating in this connection.

In the *Chronic* form, which occurs but rarely, and in which we are dealing with acute exacerbation of a chronic condition, the retention of exudate is brought about through polyps, hardened purulent secretion or cholesteatoma, which stop up the external auditory canal.

In the case of Cozzolino, to which we shall again refer, paralysis ceased gradually after the removal of the masses, while it occurred again when they accumulated anew.

Intensive Pain, Especially in the Temporoparietal Region of the Same Side.

The otitis is accompanied from the beginning by most violent pains, which are continuous, or which disappear for a few days, after the occurrence of purulent discharge from the external auditory canal. The pain is referred by the patient to the interior of the affected ear, but especially temporoparietally, and at times, also, to the depth of the orbit. The movements of the eye on the side of the affected ear may also be painful. The pain must be differentiated from that which occurs in the case of neuralgia of the fifth nerve, which is found in certain cases as an accompanying symptom in this disease. In the case of neuralgia, the pain is distributed along the branches of the fifth nerve and occurs spasmodically. In the case of the distribution spoken of, the pain is intensive and continuous. It is not influenced through antiluetic treatment, as is the case of the pain of syphilis. In contrast to the latter, it shows no greater intensity at night. At times the pain decreases and stops almost entirely, as is the case of ordinary otitis, in the first few days immediately after the appearance of purulent discharge, and does not occur again until after two or three weeks, as a forerunner of the ocular complication.

Paralysis of the Abducens.

This symptom seems to appear suddenly and unexpectedly: The patient himself notices the diplopia and calls the attention of the physician to it, who recognizes at once a paralysis or paresis of the abducens nerve on the same side as the affected ear as the cause of the diplopia. The physician could most likely recognize the gradual development of paralysis, if the appearance of paralysis could be foreseen, through a system of examination of the function of the eye muscles.

We know of cases in which the slow development of a simple pareses into paralysis could be followed. Usually, there is a recovery from the paralysis, and this also occurs gradually, at times very slowly. The measurements which have been made by Lombard in one of his cases are very instructive.

Paralysis only rarely appears a short time after the beginning of otitis. Usually it is a symptom occurring late in the disease. In 37 cases in which the time of its occurrence could be determined exactly, it occurred only three times early, that is from five to 10 days after the beginning of the otitis, 20 times after 20 to 50 days, and 10 times after 42 to 50 days. Only in exceptional cases does the paralysis occur after 56 days (in six cases).

In conclusion, one can state that it usually occurs a month or a month and a half after the beginning of acute inflammation of the ear.

It has already been said that paralysis ends in recovery in a majority of cases. Its disappearance usually proceeds gradually and slowly, at times very slowly indeed. Cases are known in which traces of paralysis were still found five months after the occurrence of the disease (Ricci). But these are only exceptional cases; as a rule, the time in which recovery occurs varies between one to three months. It appears that paralysis disappears comparatively quickly in those rare cases in which it occurs early (Baurovicz, beginning on the 5th day, disappearance on the 10th day; Tommiassi, appearing on the 7th day and disappearing on the 20th day), but this is not the rule, for there are also cases known in which an early appearance of paralysis was followed by a late recovery.

Only in exceptional cases was it observed that nystagnus preceded paralysis, or that it occurred at the same time. There are all possible graduations between paresis and paralysis.

The above mentioned characteristics mark the typical case. From the side of the eye no other symptoms can be found: the reaction of the pupils, the eyeground, the function of all the eye muscles with the exception of the external rectus are normal. The labyrinth, also, remains unaffected in the acoustic region as well as that of equilibrium. The temperature remains normal in typical cases, which are not complicated, through the entire course of the disease.

In the second category of cases, with the above mentioned

characteristics, come other symptoms which accompany the disease, or complications appear. These latter do not seem to stand in any original relation with the paralysis of the nervus abducens, because they are not present in the majority of cases, as we shall see.

The accompanying symptoms as well as these complications complete the clinical picture of the disease."

Accessory Symptoms.

The symptoms which are mentioned as appearing most frequently in typical cases, next to the three most characteristic symptoms just discussed, indicate an irritation of the meninges, or the existence of an infection in the region of the apex of the pyramid.

From this study he drew the following conclusions: There exists a typical clinical picture which is essentially characterized by an acute purulent inflammation of the middle ear and by intensive pains, especially in the temporoparietal bone region of the diseased side, and also by a paralysis of the abducens of the same side. In exceptional cases the clinical picture can be produced through acute exacerbation of a chronic purulent inflammation of the middle ear. In about half of the cases, accessory symptoms can be present, which depend on damage of the trigeminus and the oculomotorius, or of the cerebral membranes. Complications arise sometimes, such as mastoiditis and circumscribed and restricted extradural lesions on the sigmoid sinus.

Generally a complete recovery takes place, and but seldom does death result, with symptoms of a diffuse purulent leptomeningitis. The pathologicoanatomic process consists in a diffusion of the pus infection of the drum cavity to the pyramidal apex through the peritubal air cells and the carotid canal. The abducens is affected at the apex of the pyramid, close to its passage into the dura mater. We have to deal with a circumscribed osteitis at the pyramidal apex, and eventually with a corresponding pachymeningitis. The extradural location of the disease explains its generally favorable course. In individual cases, however, there occurs a circumscribed serous or diffuse purulent leptomeningitis.

"Not all cases of paralysis of the abducens, which occur in the progress of otitis, belong to the described clinical picture. Unquestioned forms of these depend on various pathologic causes. Among those occurring most frequently are the

extradural deep abscesses on the roof of the pyramid, which extend to the apex of the same. Also, a diffuse osteomyelitis of the covering of the air chamber of the temporal bone occurs, which reaches the petrous point. Cases of this kind naturally do not belong to this type, and accordingly the prognosis and therapeutics of the same appear different."

Gradenigo's studies brought forth a considerable amount of discussion pro and con, his conclusions finally being both accepted and denied. In the first few years following his original paper, case reports with discussions of the subject were not infrequently to be found in foreign journal, but American and English periodicals were notably free from contributions to the subject.

A diligent search of American literature from 1904 to 1909, inclusive, brought to light three papers reporting cases of the Gradenigo syndrome; Hastings[3], 1906; Knapp[4], 1908; the writer[5], 1909; and three others by Smith[6], Wilner[7], and Jones[8], where the association of an isolated palsy of the abducens in the presence of an otogenic infection is mentioned; although Wilner in her case ascribes the paralysis to an influenzal toxic neuritis. Eagleton[9], in discussing Smith's paper, refers to two cases occurring in his own practice. There are doubtless others to be found during this period, appearing under various titles, not indicative of this special lesion or syndrome, which were not unearthed. In 1910, Perkins'[10] classic discussion included an analysis of 95 cases, all that had appeared in publications to that date. During the past 14 years, not a few reported cases with associated observations have appeared in our own special journals, as in those abroad. However, Vogel[11], in 1921, who appends a bibliography of 129 names to his publication, gives only three American references; nor has the writer been able to find in any foreign bibliography more extended reference to the contributions from this side of the Atlantic.

The pathology underlying the Gradenigo syndrome has been extensively discussed, the major portion of authors agreeing that the anatomic position and relations of the sixth nerve at the apex of the petrous pyramid have much to do with its involvement as an otogenic complication.

Dorello's work, in making plain the details of the anatomy of the abducens at this point, is described by Gradenigo[2]. Wheeler's[12] article discusses the syndrome from the stand-

point of the ophthalmologist, and gives beautiful plates depicting the same ground. Vail[13] made dissections of 8 cadavers, and drew sketches from these illustrating admirably the formation of Dorello's canal and the various structural relations. Suffice it to note briefly here, that Dorello's canal is a variable sized, somewhat triangular shaped space at the apex of the petrous pyramid, which contains or transmits the 6th nerve, its vessels and the inferior petrosal sinus. At this point, the abducens is peculiarly liable to compression by inflammatory reaction of any type. However, as to the manner or mode in which the abducens is affected, there has been considerable difference of opinion.

Dorello[15] favors the view of transmission of the tympanic infection by way of venous channels to the inferior petrosal sinus, and thence by edema to the region of the 6th nerve with compression in Dorello's canal, the process clearing with the passing away of the aural suppuration. Alt[16], as late as 1919, insists that a reflex paralysis is the only explanation for that class of cases which give no symptoms of a deep lying inflammation other than the abducens palsy, and which otherwise appear without symptoms and disappear quickly. He has observed 3 such cases. However, he believes that cases, apart from these, for the most part are due to local inflammation at the point of the petrous.

American authorities are by no means in agreement as to an exact pathology. A local inflammation of the dura (pachymeningitis) of the Gasserian ganglion and 6th nerve is advanced by Dench[17] for his case.

This is concurred in by Shambaugh[18] and Stickney[19]. Day's[20] four cases all had pneumatic temporal bones. He ascribes the mechanism of the paralysis to an extension of the pus infection to the apical cells, the development of a circumscribed pachy- and localized serous meningitis, the latter by hydrostatic pressure affecting the function of the 6th nerve in its course through Dorello's canal. Other authors have mentioned variously serous meningitis, edema of the brain, inflammatory edema at the petrous tip, extension of inflammatory reaction by way of the superior or inferior petrosal sinus. Page ascribes to the latter mechanism the 6th palsies present in his two cases of jugular bulb thrombosis.

A number of authors believe the condition arises as a toxic neuritis. If by this is meant a specific toxin or poison passing

by means of the circulation to the 6th nerve and affecting the nerve structure itself, its etiologic relation is not so clear. This term, however, as stated by Burr[22], is used somewhat extensively, as well as loosely, where exact diagnosis is in doubt or, indeed, when no exact knowledge of the point in question exists. He points out that a toxic neuritis is usually bilateral and seldom unilateral, so that, since otogenic abducens palsy is usually monolateral and rarely bilateral, by strict interpretation of the term, toxic neuritis must be of rare occurrence.

Many observers are in accord with Gradenigo and his assumption of a definite clinical entity including the triad of symptoms and the pathologic basis brought forward by him. Quite a group, on the other hand, do not accept this pathology. On the basis of proven findings at autopsy, Gradenigo's opponents have rather the best of the argument, for there have been but few autopsies, and the findings can hardly be considered conclusive, since similar findings are met with in other cases where abducens palsy did not appear.

In so far as one can find in the reports of autopsies in these cases, no histologic study has been made of the diseased bone structure of the pyramid, the findings being purely macroscopic. This is likewise true of the sixth nerve, no report indicating microscopic study of its structure having been found.

The majority of these cases recover, and consequently, opportunity for histologic or even macroscopic study of the abducens is lacking. On the other hand, extensive operative procedures on the petrous pyramid give opportunity for macroscopic investigation of the parts reached, and these have tended to show, in a number of cases, pneumatization of the petrous pyramid to its apex, with involvement of these cells by an extension of the purulent infection from the tympanum.

When violent Gasserian pain and sixth nerve palsy are associated with this pathologic condition (and such are reported), and no other outstanding symptoms are present, the most reasonable and logical explanation is that the ganglion and nerve are directly affected from the pus process contiguous to them.

While the occurrence of the trifacial symptoms in the course of acute otitis seems to point definitely to extension and localization of the suppurative process in or at the tip of

the petrous portion of the temporal bone, an abducens paresis in the course of middle ear suppuration need not necessarily indicate an affection of the apex. It is certain that abducens paresis is often attributable to disease of the apex of the petrous portion. The abducens nerve, like the Gasserian ganglion, is in close relation with the spongy substance of the tip of the pyramid. Isolated abducens paresis is so frequently a symptom of otogenous suppurative or serous meningitis, of brain abscess and sinus thrombosis, that the arbitrary assumption of involvement of the tip of the pyramid does not seem justified. Abducens paresis in the course of otitis is a valuable sign of an affection of the tip of the pyramid only when other factors indicate such a disease. An important indication of this is trifacial neuralgia, which in itself suggests the probable involvement of this point. Abducens paresis can be referred to the tip of the pyramid only when this sign appears simultaneously[23].

The development of Gasserian pain and abducens palsy after the acute ear suppuration has apparently run its course and healed, has not been easily explained. It is well known, as Ballance[28] states: "The communication between the tympanum and the mastoid cells may become shut off by swelling of the mucous membrane, and the disease may continue to progress in the mastoid when it has already subsided in the tympanum. The writer has seen several cases of acute influenzal otitis, attended by profuse discharge, in which the disease continued to extend to the mastoid after the tympanum had healed. In one case, seen in the fourth week, the tympanum had healed, but the antrum and neighboring mastoid cells were filled with granulation tissue, becoming fibrous, while pus was found around and within the sinus groove. In another case, a boy at school, seen at the end of seven weeks from the onset of symptoms, though the tympanum had quite healed and the mastoid nearly so, pus had passed through the dura and formed a large abscess in the cerebellum."

The otitis which leads to bone disease is not always, in its initial stage, attended by any severe symptoms. "It may run its course with slight, scarcely noticeable pain, and without perforation of the tympanic membrane. In spite of the mild course, the infection may lead to an insidiously spreading inflammation, with bone destruction in any distant cell of the mastoid, which may first manifest itself, months after-

wards, by general symptoms and external mastoid signs. The locus of disease may remain completely shut off from the tympanum, or the tympanum may be reinfected from the disease in the bone. A serous exudation is sometimes found in it, in addition to the pus in the bone."

Acute suppurations of the tympanum, when not properly relieved by incision of the tympanic membrane, may give rise to osteomyelitis of the spongy portions of the petrous bone (the bone immediately surrounding the labyrinth, and in the vicinity of the bony portion of the Eustachian tube). When this takes place, the patient is in the greatest danger[26].

Körner[27] reported the experience in a patient who was operated on too late and who died from purulent meningitis. There was disease in the spongiosa in the petrous, as well as extensive destruction of mastoid. This condition can hardly be eradicated at an operation, unless the operator is prepared to sacrifice the labyrinth and the facial nerve.

With these facts in view, the class of cases mentioned above seem somewhat analogous to this socalled latent mastoiditis, but with an exact reversal of the process. That is to say, the aditus from the beginning becomes impervious to purulent transfer from the tympanum to the antrum and mastoid cells; while the infection extends to the apex through the cells of a largely pneumatized pyramid, but, being slow and insidious in its progress, the tympanic infection heals and later may be reinfected from the infection at the apex. These are the cases which show little or no mastoid involvement when operated upon.

In the slow development, vitally important factors are: 1. The pathogenic organism. 2. Favorable anatomic structure of the temporal bone. 3. Virulence of the organism. 4. Resistance of the patient.

Time is lacking to go farther than the few points already touched upon. Three cases illustrating three different phases of otogenic disease associated with paralysis of the abducens are added below. The writer is much indebted to Drs. Herbert M. Goddard and Matthew S. Ersner for permission to report cases two and three.

Case 1: November 29, 1907, the writer was called to see T. F. B., white, male, age 30, who gave a history of very severe pain affecting the left side of the head for the four preceding days. Temperature 99.3°, pulse 90, respiration 20.

Prior to the onset of the pain, which was agonizing at times, he had suffered from a slight head cold. The cold, however, had cleared, and at this time gave no symptom. It was noteworthy that at no time had there been any pain whatever in the ear, and, although impairment of hearing was marked, the patient had not noted it until his attention was called by examination. Hearing was reduced to C. V. at 10 feet, Weber to the left; Schwabach lengthened, and Rinne negative.

Inspection showed an approximately normal external canal. The tympanic membrane, however, was dull, thickened, and showed marked bulging. Incision of the membrane was followed by free purulent discharge. The pain was relieved by the flow of pus, which continue free for three days, when pain again became severe, coincident with a decrease in the quantity of purulent discharge. A second incision of the tympanic membrane became necessary 12 hours later. Again, the purulent flow was quite free, for three days, and the patient was free of pain. This was followed by a considerable decrease in the amount of pus, and an immediate increase of pain. The membrane was again incised, with an increased flow of pus and similar easing of pain. At this time, there was some mastoid tenderness, which gradually decreased and passed away in several days. Temperature during this period had varied from 99° to 100°.

The purulent discharge was very free for another week, and then began to gradually lessen, but without return of pain. On December 17th, the patient complained of double vision. Examination showed homomynous diplopia, the separation of images increased on looking to the left—a paresis of the left externus; T. P. R. at this time normal.

The next morning the paralysis was complete. This was shown by a marked convergent squint, accompanied by annoying diplopia and vertigo. The vertigo was promptly relieved by closure of the affected eye, thus indicating that it was due to diplopia and muscle paralysis, and not to an intracranial complication. Further, the left eye could not be rotated externally beyond the midline. An added complication, occurring simultaneously with the complete palsy of the muscle, was a polyarthritis, accompanied by a rise in temperature.

Examination of Eyes: Movement of the lids of both eyes normal; considerable injection of the conjunctiva of

the left eyeball. Pupil of each eye, 2.5 mm., reacted freely and equally to L. and C. The media were clear, discs clearly outlined, and no evidence of papilledema or gross lesion of the fundi, V = 20/15 — 2.

Ocular movements were unaffected except in the field of the left externus. Pain was elicited by pressure over the area of the affected muscle and by ocular movement, especially by a strong effort at rotation toward the affected side. Bacteriologic examination at this time showed streptococci and staphylococci, streptococci predominating. No lumbar puncture was done or X-ray study made at this time. A skiagram taken recently revealed a pneumatic mastoid of very extensive development.

The purulent discharge ceased within ten days, and the arthritis improved more rapidly than the palsy. By January 18, 1908, no diplopia could be elicited by extended ocular rotation to the left, so that all symptoms of the paralysis had cleared. There has been no recurrence.

Comment: 1. The onset of severe left head pain was the first symptom to be noted. The pain was relieved by drainage of pus and recurred with retention.

2. The abducens palsy appeared three weeks after the onset of the otitis, when the disease was on the wane, and it finally disappeared thirty-two days after its appearance.

3. The complicating polyarthritis arose coincidently with the palsy, but healed at least two weeks earlier.

4. There were definite signs of local inflammatory reaction about the affected muscle.

CASE 2: (Courtesy of Dr. H. M. Goddard.) C. R., male, white, age sixteen years. Chief complaint, pain and loss of hearing in right ear. Past medical history negative, except for influenza during the recent epidemic, and recent night sweats, loss of weight, and strength.

History of Present Illness: August 2, 1924, onset of acute otitis with high fever, the sequence of a sore throat which had healed in one week before. The following day spontaneous discharge occurred, and two days later he noted that he was entirely deaf.

Physical examination, August 14, 1924, on admission to the hospital, negative, except for discharge from the ear. The tympanic membrane showed a red polypoid mass at its posterior superior aspect, a small perforation beneath, canal nor-

mal. Nasal examination negative. August 14th, culture from throat showed pneumococci staphylococci and streptococci. Culture of pus from right ear showed staphylococcus albus. August 21st, the discharge had almost ceased. At this time a complete sixth nerve paralysis, associated wtih a severe periorbital and suboccipital neuralgia developed. The eye-grounds were negative. X-ray of mastoid practically negative. Neurologic examination negative, except for sixth nerve palsy.

August 27th, a simple mastoidectomy was performed, and very little pus found in the antrum and subantral cells. Polypoid mass of the tympanic membrane removed. Following the operation the pain decreased, the ear drained profusely at first, later decreased, but the paralysis remained constant. On September 3rd, twelve days after the mastoidectomy, the patient had the first symptoms of a fulminating meningitis, which resulted in his death twenty-six hours later. Autopsy was refused. He ran a light evening temperature throughout his illness until meningitis developed. Pulse rate, average 72; respiration 20. Urine was negative throughout. Blood count; red cells, 4,000,000; white cells, 13,900; polys, 74%.

Case 3: (Courtesy of Dr. M. S. Ersner.) H. W., age 17 months. Acute Bilateral Mastoiditis, March 13, 1924.

Patient entered hospital on March 13, 1924. Family and previous history negative. Present condition started several weeks ago with pain and discharge from both ears. Mother also noticed simultaneously that one eye turned in.

Examination: Right: (1) Profuse creamy discharge. (2) Sagging of posterior superior canal wall assuming nipple like protrusion. (3) Folds more prominent and slightly thickened. Slight protrusion of auricle.

Left: (1). Profuse purulent creamy discharge. (2) Sagging of posterior superior canal wall. (3) Folds slightly lifted with lower fold smoothed out. (4) Urine: negative. Occasional leucocytes and squamous epithelial cells. (5) Blood: 3,650,000; 10,100; 68% hemog.; 65 small lymph; 35 polym; 1 eosin. (6) X-Ray: No appreciable difference between the right and left mastoid processes. Left side looks definitely normal. The semicircular canals clearly visualized. There is a faint suspicion of infection in the posterior cells on the right side. This evidence is not sufficient to be regarded with much weight. (7) Eye: Slight veiling of the edges of the discs.

Apparent weakness of the left external rectus muscle, March 13th. March 16th, no evidence of intracranial pressure.

Operation: March 15th. Blood clot closure with drain. Right side: (1) Periosteum stripped easily except at the bottom. (2) Cortex slightly roughened and found thick. (3) Cavity under the cortex filled with free pus. (4) Involvement of the zygomatic cells. Culture: Streptococcus.

Left side: (1) Periosteum stripped easily. (2) Cortex moderately thick. (3) Cavity under cortex filled with free pus. Culture: No growth.

Postoperative Period: March 18th, removed stitches and inserted gauze drain. Drainage abundant. March 19th, wounds draining freely, also considerable discharge from ear. March 20th, right wound wide open, left wound coated in upper half. Good drainage. March 27th, temperature normal, wounds granulated and nearly closed, only slight drainage. Both ears dry. Drums assuming normal greyish color. Weakness of external rectus disappeared and no strabismus can now be detected. Discharged to be cared for by family physician.

REFERENCES.

1. Gradenigo. Arch für Ohrenheilk, 1904, vol. LXXII.
2. Gradenigo. Ibid, 1907, vol. LXXIV, p. 179.
3. Hastings, Hill. Arch. of Otology, 1906.
4. Knapp, Arnold. Arch. Opth., vol. XXXVIII, p. 552.
5. Sears, Wm. H. Penna. Medical Journal, Aug., 1910.
6. Smith, E. Terry. Otological Soc., 1905-07, vols. IX-X, p. 551.
7. Wilner, Anna S. Archives Pediatrics, Jan., 1907, p. 18.
8. Jones, Clarence P. Laryngoscope, July, 1908.
9. Eagleton, Wells. Otological Soc., 1905, vols. IX-X, p. 553.
10. Perkins, Chas. E. Annals of Otology, etc., 1910, p. 694.
11. Vogel, Herbert. Internat. Zentral. f. Ohrenheilk., Aug., 1921, Bd. 19, Heft 1.
12. Wheeler, John M. Transactions Sect., Oph., A. M. A., 1918.
13. Vail, H. H. Larnygoscope, Aug., 1922, p. 569.
15. Dorello. Monatsch. für Ohrenheilk., Bd. XLVI, p. 332.
16. Alt, F. Ibid, 1919, Bd. LIII, p. 534.
17. Dench, E. B. Annals of Otology, etc., Sept., 1916, p. 672.
18. Shambaugh, Geo. E. Personal communication.
19. Stickney, Otis. Laryngoscope, 1919, vol. XXIX, No. 7.
20. Day, Ewing. Atlantic Med. Journal, June, 1924, p. 572.
21. Page, Jno. R. Annals of Otology, etc., June, 1916, p. 497.
22. Burr, Chas. W. Philadelphia, Pa. Personal communication.
23. Schlander, E. Monatschs. für Ohrenheilk., etc., Vienna, Bd. LVII, p. 975.
24. Ballance, Chas. A. Surgery of the Temporal Bone, vol. I, p. 107.
25. Körner. Die eiterigen Erkrankungen des Schläfenbeins, 1899, p. 29.
26. Haberman. Arch. für Ohrenheilk., vol. XLII, p. 128.
27. Körner. Loc. cit., p. 32.

CASE REPORTS.

1. W. H. Sears, Huntingdon, Pa.
2. H. M. Goddard, Philadelphia, Pa.
3. M. S. Ersner, Philadelphia, Pa.

DISCUSSION.

DR. GEORGE F. KEIPER, Lafayette, Ind.: Permit me again to call your attention to the condition which I have chosen to call "Vacuum Mastoiditis," where pus does not issue from the middle ear, and no pus is found in the mastoid process.

Several years ago, before the advent of X-ray diagnosis, I was consulted by a lady, Mrs. F, from an adjoining town, who suffered with almost all the classical symptoms of acute mastoiditis. She suffered terrifically from pain, and the mastoid was very tender to touch and pressure. I took the usual precautions to distinguish between psychic and organic pain. Her pain was organic, but on opening the mastoid, no pus was found, and the lining membrane of the cells was very hyperemic. The bone chips revealed nothing in particular. To say the least, I was very much chagrined, and her physician was of the opinion that an unnecessary operation had been performed. For quite a while my conscience troubled me, until Dr. Greenfield Sluder called our attention to what he called "Vacuum Headache," which yielded to nothing but reopening the frontonasal duct. Then a great light dawned and I saw that a similar condition had happened to this woman's aditus ad antrum.

Another patient, Mrs. Rose G., with similar symptoms, appeared later on. X-ray plates revealed no pathology. Her temperature was normal, her pulse was 90, and her respiration 30. She was a victim of migrain. Two years before, she underwent a successful operation for acute appendicitis. All laboratory examinations were negative. I recommended opening the mastoid, to replace from without the air which had been absorbed from within, and confidently told her physician that no pus would be found, but that aeration of the mastoid was necessary for relief. No pus was found. The bone chips revealed nothing. The wound was sewed up tight after a thorough examination of the mastoid. All pain ceased immediately, never to return.

The last case of this description I saw was six months ago. Charles D. came suffering with a left mastoiditis, but with no discharge from the middle ear, and with negative X-ray plates and laboratory findings. All remedies used to alleviate pain were futile. The usual pain tests revealed organic pain. His history revealed, that while inspecting a locomotive four months before, he fell on alighting and struck the right mastoid process. For a month there was tenderness, of course. It continued mild until a week before he first saw me, February 25th. During that week, he suffered terrifically from pain in and over the mastoid. An examination revealed a retracted ear drum with a stenosed Eustachian tube.

Here was an interesting situation as to compensation under our laws. Was the condition due to the accident? Was the railway liable? Of course, I predicted to the chief surgeon that no pus would be found, but if the aditus ad antrum was occluded by bony formation, the liability would be with the railroad; if with an inflammatory thickening of the aditus, the liability was with the patient. I asked the local surgeon to assist me in the operation. Ae we uncapped the mastoid cells, he remarked that he could see the air bubbling through the blood overlying the operative field. After thorough examination, we explored the aditus

ad antrum and found the occlusion to be bony. We could not enter the middle ear, and we did not persist in our efforts to do this. The liability was with the railway company. He made a prompt recovery.

Dr. Joseph C. Beck, Chicago: I wish I could be as certain of the diagnosis and indications for a mastoid operation as Doctor Hayden. He states that it is time we should say definitely when mastoids should be operated upon. I am not ready to say that yet, irrespective of all the statements that Doctor Hayden has made.. He has completely and absolutely ignored the pathologic changes that are recognized in various types of mastoid disease as an indication for operation, and while we cannot depend upon that for diagnosis of mastoid operations, we should have a clear knowledge of the various types of pathologic changes present which produce certain manifestation that would be an indication.

I was asked by the last essayist to discuss the Gradenigo syndrome and the value of it. I have had only two cases wherein this syndrome was very definite, but one part is severe pain. That pain is excruciating; it is Gasserian ganglion pain, in connection with paresis and followed by paralysis. In one of my cases, there was also a symptom that the doctor did not mention, and that was a labyrinth choke, a deafness, in which there was no vestibular reaction. This subsequently cleared up somewhat, so that patient heard and had a vestibular reaction.

Dr. J. J. Sullivan, Jr., Scranton, Pa.: Dr. Sears requested me to report the following case:

Adult, male, which resembled Gradenigo syndrome in some essentials. This patient had had two operations on his mastoid for a suppurative condition, but still suffered from severe temporal pain and facial neuralgia. The discharge from the middle ear was still fairly profuse. There was a small portion of the tympanic membrane remaining. The radical procedure was instituted. About the time of completion, he had a complete "acute" exophthalmus, with deviation of the eyes inward. Patient became cyanosed. I noticed a small amount of pus behind the external semicircular canal. On probing, pus came out freely; the depth of the cavity was ¾-inch into deep petrous cell. All symptoms improved on enlarging the opening, except the "exophthalmus," which persisted one week. The wound was left open, and a small drain was inserted into the deep petrosal cell. All pain disappeared within 24 or 48 hours. A facial paralysis appeared in five days. This has slowly improved. Discharge persisted for one month after operation, at which time we made the auricular flap.

Dr. Albert E. Bulson, Jr., Fort Wayne, Indiana: There are no hard and fast rules that can be formulated covering the necessity for operation for acute mastoiditis, and I do not believe that we ever will be able to lay down any such rules. Every case is a rule unto itself, and the more experience we have, the more we are guided by the variety and character of manifestations of the composite picture in deciding upon operative measures. In my judgment, acute mastoiditis is operated too often unnecessarily, and in the practice of conscientious operators, this is due to the misinterpretation of the indications of certain symptoms or manifestations. Mastoid tenderness is one of the symptoms that all too frequently leads to operative interference, especially in the practices of the inexperienced, who may fail to take into consideration other

factors in the case. One must remember that tenderness is present as an early symptom in a large percentage of the cases of acute suppurative otitis media that go on to recovery without surgery of the mastoid. Tenderness of the mastoid, therefore, is only a part of the syndrome that must be considered. This also is true of pain in the mastoid, which is an indifferent manifestation, varying in its character and intensity, and oftentimes not present at all. Elevation of temperature is another inconstant and varying manifestation, which depends upon the nature of the infection, character of drainage, and lowered resisting power. Even the differential blood count, unless especially significant, must not be taken too seriously, unless coupled with other manifestations. Redness and swelling of the posterior superior wall of the meatus perhaps may be considered pathognomonic and indicative of the necessity of operative interference on the mastoid, but on the whole, it is the composite picture which is considered by the experienced operator, and discriminating care must be exercised in determining all of the varying manifestations and analyzing them in the light of previous experience. Fulminating cases of mastoiditis, occurring within one or two days after the onset of middle ear symptoms in what previously was a perfectly normal ear, are rare, and require prompt attention; but they, too, present the composite picture which leads to the very proper decision to operate.

On the whole, the mastoid operations performed anywhere from one to three days after the onset of middle ear symptoms, so often a feature in the practice of some men, should be condemned. I believe that nearly all cases of marked inflammation of the middle ear cavity are accompanied by congestion of the mastoid antrum, through contiguity as well as continuity of structures, and that the tendency is toward recovery rather than the development of suppuration, providing the condition is recognized promptly and suitable treatment instituted. This means very early and free drainage of the middle ear cavity by a wide incision in the drum membrane. Operative interference in the acute inflammations of the middle ear cavity will, to a large extent, do away with the necessity for mastoid operations.

Dr. Samuel Salinger, Chicago: I want to say a few words with reference to pain. There are some cases, and we have had three in the last year, in which you have pain that is all out of proportion to the other symptoms, pain that is excruciating, that lasts half the night, and is not relieved by opiates. This sort of pain drives the patient frantic, and very often leads to premature and unnecessary operation. On consulting neurologists, I find that some cases are due to neuralgia of some of the contributing branches of the otic ganglion. This was well brought out in an interesting work by Ramsay Hunt some few years ago. I think we ought to take cognizance of the fact, that severe pain, out of proportion to the inflammatory symptoms, is an indication that we may forego operation.

Dr. J. J. King, New York City: I wish to emphasize one point in Dr. Hayden's paper. If I heard correctly, he did not emphasize sufficiently the importance of the streptococcus mucosus capsulatus in the consideration of otitis media for operation. There are many who feel that simply the finding of this organism is sufficient to indicate operation,

but in my opinion, there should be serious consideration given to the other symptoms which he so completely presented.

In the discussion of Dr. Sears' paper, I wish to state, that I believe that paralysis of these nerves and muscles is more likely due to toxemia than to the direct extension of the infection to the areas from which they take their origin. I had a case last year which presented paralysis of the external rectus following mastoid and acute exophthalmos, an old mastoid operated five or six years ago. I did an immediate operation on this child, and she had a small mastoid cavity with complete sinus thrombosis and a localized meningitis, which was very visible when we got down to the dura. I did a mastoid, ligated the jugular, and exenterated the sinus, and all the symptoms, including the choked disc, cleared up in a reasonable length of time, and the patient made a good recovery. I believe if this had been a direct extension of the infection, this patient would have died.

Dr. Frank L. Dennis, Colorado Springs, Colorado: Owing to the infrequency of the Gradenigo syndrome, I simply want to call attention to the pathologic report of a case I had this last summer. This man had acute otitis with excruciating pain, which, however, was not confined at all to the mastoid. His pain was mostly in the distribution of the fifth nerve of the face, and was so intense that opiates were required, but even they did not relieve him. He had a mastoid operation, and considerable trouble was found there. For a week, he apparently progressed favorably, then he developed meningitis and died. He had two of the triad of the Gradenigo syndrome, but not the paralysis of the external rectus. However, the interesting part of the case was the postmortem findings. The report was that the cells in the tip contained pus, but the overlying part was not pneumatized. We found at the tip of the petrous pyramid, under the depression for the Gasserian ganglion, a fairly large cell which contained pus. Sections were made of this, and the report was, that in the right Gasserian ganglion there was marked purulent inflammation, which undoubtedly was the cause of the pain. This man died in two weeks after the operation, and in view of what Dr. Sears has told us about the length of time it takes for these things to develop, I think it is very likely, that had the man not had his meningitis, he probably would have developed a typical Gradenigo syndrome.

Dr. L. E. White, Boston, Mass.: Pain in the acute stages, with an unruptured drum membrane, is expected. If the ear is discharging freely, ordinarily the pain is relieved. The presence of pain with an open ear drum and free discharge, indicates that there is too much secretion being formed within the mastoid to find exit through the aditus. An interesting case occurred in my last service at the infirmary. An Armenian woman came into the clinic complaining of very severe pain. Under the usual treatment of free incision, the use of ice over the mastoid and general treatment, the pain subsided and the discharge nearly ceased, and needing beds, we let her go from the hospital. She returned in a week appearing to be very sick. The pain had returned. Opening of the mastoid to relieve that pain. She was operated on the second time, to see if some portion had been overlooked. In spite of that, she developed meningitis. On autopsy, we found the infection had extended from the zygomatic region to the temporosphenoidal region. The pres-

ence of pain after the mastoid is opened, recurring after the ear has apparently subsided, is, I think, a very valuable indication that there is something going on that has not been reached.

Dr. William H. Sears, Huntingdon, Pa. (closing): With regard to the question as to when a radical mastoidectomy might be indicated, rather than a simple one: Naturally, if there has been a preceding chronic otitis, a radical mastoidectomy would be necessary, but in acute otitis, after a simple operation has been completed, sometimes there will be an extensive purulent discharge from the external canal, and practically none from the mastoid wound. In two cases of my own and a great many of those reported, the purulent discharge from the external canal has been so profuse, that it could not possibly arise in the tympanic cavity, but must necessarily have come from a group of adjacent and communicating cells. In this group of cases, where, after a simple mastoidectomy, the other symptoms have not been relieved, a radical procedure is indicated.

The case which Dr. Beck reports is one of the unusual types. Gradenigo states that affection of the labyrinth does not occur in his typical group, and is but rarely associated at all. However, Hill Hastings, in 1906, reported the first case of the Gradenigo syndrome which appeared, so far as I have been able to find, in American literature. This case had an associated suppurative labyrinthitis for which he did a labyrinthectomy, and the patient made a complete recovery.

Dr. King assigns the cause of the abducens paralysis in his very interesting case to a toxic neuritis. Study of the literature indicates that this syndrome can arise by different etiologic routes. As mentioned before, Alt gives a series of otogenic palsies based on varying etiologic factors. Among these he includes toxic neuritis. But all cases are not due to toxic neuritis, otitic meningitis, extradural abscess, cerebral edema, or to the pathology proposed by Gradenigo. Unfortunately, of those cases with a lethal ending, but few have come to autopsy, and in these autopsies very little attention and record has been given to pathology of the petrous pyramid.

THE TREATMENT OF PARANASAL SINUS DISEASE IN CHILDREN.

MILLARD F. ARBUCKLE, M.D.

ST. LOUIS, MO.

When I was invited to read a paper on this subject, I decided to describe our methods of working, and to present the clinical record of a few typical cases. There are, I am sure, possibilities for improvement in our methods, and in order to emphasize these possibilities, I hope the members will be generous in their criticisms and suggestions during the discussion.

Before considering the matter of treatment, I wish to again lay emphasis on the fact, that very frequently, there is an etiologic relationship between suppurative disease of the nasal sinuses and various types of constitutional disease, which are of infectious origin. It is my feeling, that inflammatory disease of the upper nasal cells is responsible for constitutional disease much more frequently than we have formerly supposed it to be. This is demonstrated in numerous cases of heart disease, rheumatic fever, multiple infectious arthritis, nephritis, chorea, lymphadenitis, anemia, malnutrition, optic neuritis, otitis, mastoiditis, etc.

The necessity for close cooperation between the rhinologist and the pediatrician, the neurologist, the orthopedist, and the ophthalmologist, is obvious, when it is realized that on the rhinologist falls the burden of finding the cause and providing relief therefor in diseases of such grave import as above mentioned. Symptoms and signs such as loss of weight or underweight, lack of appetite and energy, anemia, daily low grade fever, enlarged glands in the posterior triangle of the neck in association with one or more of the above mentioned disorders are highly suggestive of suppurative sinusitis. In many cases, the X-ray findings of the chest and the symptoms resemble those of tuberculosis so closely, that this diagnosis is not infrequently given. Study of the nose by posterior rhinoscopy and with proper light is imperative in the diagnosis and treatment of these cases.

After the diagnosis has been established, we are confronted with the task of cleaning up the infection with the minimal danger and inconvenience to the patient and his family. Experience has taught us, that in most cases it is possible, by proper hygienic and dietetic measures, in addition to correct local treatment, to cure infections of the nasal mucosa in children without operation. Occasionally, however, we see cases of long standing and well established suppuration within the sinuses, which will respond to treatment only after the sinuses have been freely opened so as to admit of more perfect ventilation and drainage, as well as the application of bactericidal agents to their interior. It is in this sort of case, usually, that we see the severe constitutional disturbances already referred to. The results of such treatment are gratifying both in so far as the local as well as the general condition is concerned. Occasionally it becomes necessary to reopen or to enlarge the opening into the sinuses, and when necessary this should be done without hesitation.

The advantages of pure country air over that of crowded city quarters are very great; also proper food is of great importance. Cod liver oil is an important remedy, and one which nearly always is indicated. The bactericidal remedies with which we think we have secured the most satisfactory results have been mercurochrome, gentian violet and acriviolet. The latter is said to be more efficacious in an alkalin field, but in my experience the alkalin solution has been the cause for complaint more often than has the acriviolet.

If the nose is filled with pus, this should be washed out with a warm saline irrigation. There is of course always danger of otitis in the presence of pus in the nose, and this danger is increased by attempts at removal. This danger is greatly reduced, however, if the child lies face downward during the irrigation, with head over the edge of a table, so that the irrigating fluid runs gently up into the nose and down again, carrying with it the pus.

I have never seen an ear infected where this method was followed. After the irrigation has been completed, and the fluid has all run out of the nose, the child should lie face upward, and with the head over the edge of the table, a few drops of the medicine is then allowed to run high up into the nose and back along the cribriform plate to the olfactory fissure, before the child gets up. One or two treatments per

day, according to conditions and results, will be required at first. In cases without large amounts of pus the irrigation is not required.

When maxillary sinus suppuration is present, irrigation with the trocar, in addition to local treatment, often is followed by complete recovery. The use of negative pressure has been somewhat disappointing to me.

When drainage by surgical interference is indicated, it is our practice to open the sinuses under nitrous oxid anesthesia. Preoperative treatment is resumed after about eight or ten days. The result of surgical drainage will be demonstrated in some of the slides to be shown.

As will be seen by the case reports, the treatment often must be continued over a rather long period. It is of course desirable to shorten this period as much as possible, and suggestions to that end will be appreciated. Another point which I wish to emphasize is the certainty of results and the comparatively short time in which improvement will be noticed.

Case Reports.

Guy S., age 4½ years, was admitted to Children's Hospital July 26, 1923, because of swelling of feet, ankles, abdomen and face, which began about July 15th. There was no history of cold or sore throat. He appeared puffy about eyes, and then other edema was noted and steadily increased. He had been put on a milk diet before entrance, but had not improved. The family and past history were negative.

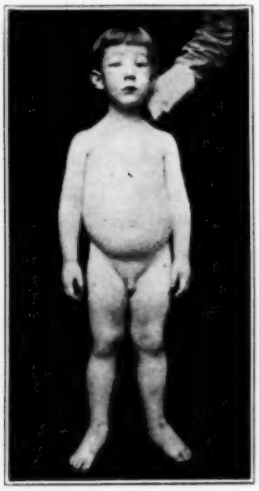
The striking thing, of course, was the tremendous edema of the whole body, with no pain and no subjective distress except interference with vision by swollen lids. There was pitting edema of trunk, face and extremities, and free fluid in the abdomen. Heart and lungs normal. Tonsils were moderately enlarged and ragged but showed no acute inflammation. The urine was scanty in amount, 500 c.c. a day, with large amounts of albumin. W.B.C. 10,000. Blood W. negative. Nonprotein nitrogen 60 mgm. Temperature 101 deg. on entrance, and averaged 99.4 after.

Course: He was given usual nephritic care and soon began to improve, weight began to fall, and edema left the face and then extremities. B. P. always normal. Tonsils and adenoids were removed August 7th, and recovery was uneventful. On August 25th, antra were irrigated and pus

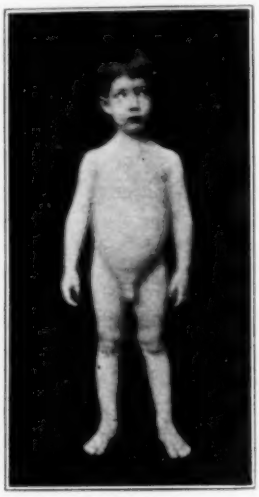
found. He was dicharged August 27, 1923, free of edema, and urine showing moderate amounts of albumin.

From August 27th to Sept. 16th he was well. On Sept. 17, 1923, he caught a "head cold", and generalized edema promptly returned. As before, urine output in the acute stage was scanty. He was admitted afebrile, and most of the course was so.

The sinuses, antra, ethmoid and sphenoid were found infected and were drained. Staphylococcus aureus was the organism as in previous admission. The antra were very



Case 1. 7-31-23.

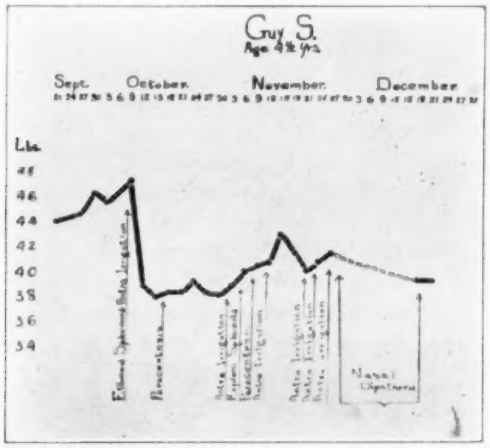


Case 1. 8-27-23.

difficult to keep open and draining freely, and were repeatedly punctured. To add to his troubles, he got a ward infection of diphtheria. By December 17th, he was free of B. diphtheria. His edema had passed, his appetite and strength were rapidly picking up. The nasal discharge was still present but not very marked. He was sent home December 21, 1923, to be followed in Out Patient Department. The mother was taught how to irrigate his nose, and given mercurochrome solution to use afterwards.

On Jan. 2, 1924, he was again admitted with temperature of 104 degrees, and the history of an acute gastorintestinal upset, with much diarrhea, of about two days duration. This infection was never localized further and temperature speedily returned to normal. There was no increase in infection in

nose; in fact it seemed clearer than ever before. There was no increase in weight during this admission, and no edema to be found. The boy weighed 37½ lbs. on admission and 35 on discharge. He was given two transfusions on this admission to improve general strength. Eyegrounds showed no abnormal findings. He was discharged January 10, 1924,



Case 1.

Case 1. 2-14-24.

Case 1. 7-1-24.

apparently a normal, hungry boy again. The nasal infection improved, and the boy gained in weight.

About April 1, 1924, he began to have fever and diarrhea, and in the course of three days was critically ill. Temperature was 104; he was quite prostrated and whole abdomen was painful. WBC was 50,000. No pneumonia could be found. Blood culture was sterile. By April 10th, there was a definite cellulitis of right calf of leg, which was incised, and hemolytic streptococci were found in the small amount of pus obtained. The boy rapidly improved, to have a similar occurrence in left calf ten days later, which was also drained. Meantime there was no edema, and only a little pus to be seen in the nose. The leg wounds healed speedily. The boy was sent to the country farm, where, with the care given, he gained 10 pounds in three weeks, and was discharged June 10th in very good condition. Urine on discharge showed only a heavy trace of albumin. The boy has been seen since and weighs 45 lbs. Now 5½ years old. Nose is clean of obvious infection. Urine shows only faint trace of albumin.

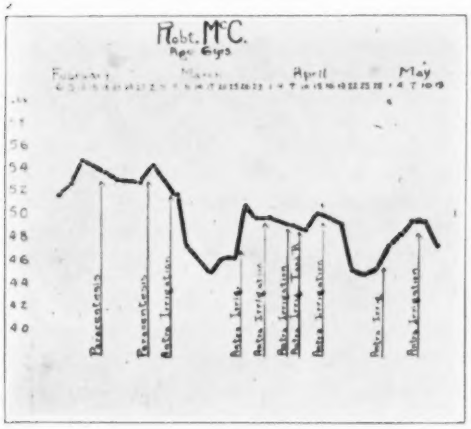
Robt. McC., age 6 years, admitted Feb. 5, 1922: Past history negative before onset of present illness in December, 1921, when the boy had a severe head cold with profuse purulent nasal discharge. There was no sore throat. Some two weeks after onset of "cold", edema of face was found, and the urine examination showed much albumin. He was put on milk diet and few vegetables, with some improvement in generalized swelling. About Feb. 1, 1922, abdomen began to swell and face became puffy.

The boy was quite edematous with pale, pasty skin and a puffy face. Tonsils were large and ragged, but there was no redness or exudate. There was purulent nasal discharge. The heart and lungs were negative. B.P. 110-70. Abdomen was distended with free fluid, and there was slight edema of scrotum and extremities. Temp. 99 degrees, weight 52 lbs., and 55 lbs. five days later. Urine contained much albumin and numerous casts, and was reduced in amount. Nonprotein nitrogen, 45 mgms. per 100 c.c.

This patient was in the hospital until May 13, 1922. During this time, he had ten irrigations of maxillary sinuses, the first being February 28th. The culture of pus present was always staphylococcus aureus. He had otitis media a time or two. On April 7th, tonsils and adenoids were removed. The same feature was noticed time after time—when the nose

showed any amount of pus, the edema was easily seen; and with irrigation of antra and removal of pus, the boy would lose 2 or 3 pounds in the next week. Numerous times his eyelids and face were puffy, and there was some fever (99-101 degrees) and urine was heavy with albumin and casts, while

Case 2. 4-4-22.



Case 2.

nasal examination or irrigation of antra would reveal a lot of pus. The boy's nose was irrigated with saline solution when any amount of pus was to be seen. Diet contained sufficient protein for demands, but was mainly carbohydrate, the protein generally from milk.

On discharge the boy weighed 48 lbs. and was free from edema, though the urine contained small amounts of albumin. The sinus irrigations no longer contained pus. Color was

good, and it was probable the boy had gained weight that was not all fluid. In April, 1924, this patient's family physician reported that the boy had continued well, and urine was normal.

Helen L., age 7 years: She was admitted Sept. 19, 1922, with a moderately severe chorea, which had continued more or less constantly since January, 1921. She had been subject to frequent colds, and in early January tonsils and adenoids were removed. The chorea came on about 3 weeks later. Besides the chorea, which prevented dressing or feeding herself, the patient was poorly nourished and very unstable emotionally. She had a slight chronic seropurulent nasal discharge.

She was sent to the Country Farm to build up nutrition. Her weight went up to 50 lbs., and the chorea seemed less marked. Heart action continued rapid.

In February, 1923, she was sent back to hospital because of profuse purulent nasal discharge. The antra were irrigated and considerable thick pus washed out. She was sent home for two weeks and then returned, and the nasal infection was vigorously treated. Chorea was ever present, and had never completely cleared. She remained under care from March to the middle of October, 1923. She would have her sinuses washed several times, and then be sent back to country, where local treatment and irrigations were done, and then return for another round. In all, both antra were washed out eleven times, and each time gas oxygen anesthesia was used. Both sphenoid and ethmoid sinuses were drained.

Early in the summer, the patient developed a heart lesion that had not been present before. By October she had lost most of chorea—only an occasional jerk—was much brighter, and nearer a normal girl; color was good and nutrition excellent. She returned to school, got along normally and grew as she should. In May, 1924, she returned with an acute otitis media, which cleared up after puncture of drum. Her nutrition was much better. Chorea had passed. She weighed 56 pounds. The sinuses showed no obvious infection, and more astounding still, the heart murmurs had disappeared and the cardiac action was normal. The girl has remained well and carries on as a normal girl.

BIBLIOGRAPHY.

1. From the Department of Oto-Laryngology, Washington University School of Medicine.
2. Dean, L. W. Paranasal Sinus Disease in Children. University of Iowa Studies in Medicine, April 15, 1921.
3. Dean, L. W. Treatment of Paranasal Sinus Disease in Infants and Young Children. Laryngoscope, Jan., 1924, vol. xxxiv, pp. 30-35.
4. Arbuckle, M. F. Systemic Manifestations of Suppurative Disease in Paranasal Sinuses. Jour. A. M. A., Sept. 1, 1923, vol. lxxxi, pp. 741-743.
5. Arbuckle, M. F. Diagnosis and Treatment of Inflammatory Disease of the Nasal Accessory Sinuses. Sou. Med. Jour., April, 1924, vol. xvii, pp. 293-297.

DISCUSSION.

Dr. John J. Shea, Memphis, Tenn.: I differ from the essayist as to the value of gas as an anesthetic of choice in sinus surgery. It is preferable for the exploratory punctures, but if the patient's condition warrants the use of ether, this latter anesthetic given with the continuous flow is a better anesthetic, as it allows of more careful surgical procedures.

The following conditions in children have required surgical interference on the sinuses by me during the past year: Asthma, 7; cyclic vomiting, 1; pyelitis, 5; persistent cough, 3; acute myocarditis, 2; acute nephritis, 3; meningeal symptoms from an acute sphenoidal suppuration, 1; hypertrophy of lingual tonsils, 1; orbital abscess following a ruptured frontal sinus, 1. All of these cases except the last had double antrotomies, while the last required a Lynch radical frontal operation.

The operation was practically the same as that done by Dr. Arbuckle, including the use of his antrum knife, but with the addition of inserting a rubber tube through the antral window into the sinus for continuous drainage and irrigation. It is necessary to irrigate these cases until they are free from purulent discharge. As long as the tube remains in place, you will have some mucopurulent discharge from the irritation of a foreign body in the sinuses. At times, we have retained the tube in place for a week, but the average has been four days. The tube serves the double purpose of creating a semipermanent opening for future irrigation as well as for the present treatment. In some of these cases, I have irrigated the antra as often as three times a week for six or eight months, while in the asthma cases we have irrigated them only whenever they present prodromal symptoms of asthma or to clear up an acute infection. Likewise, in the kidney cases, we reinstate the irrigations upon the onset of any symptoms. We have taken radiograms of these cases at six month intervals, and we believe that the growth of the sinuses as shown by these plates is the best indication as to the progress of the treatment. In the majority of the chronic conditions for which we operate, the sinuses were morphologically of the arrested type. In fact, we consider the information obtained as to the stage of the development as the most reliable indication that the sinuses have entered into the etiology of the patient's pathology.

Case 1. H. K., aged 7, first seen May 16th, 1922. Chief complaint, asthma, for which patient has had tonsils removed and the tests made for protein sesitization, with no result. Upon examination, the nasal

mucous membrane was of the vasomotor type and the middle turbinates were swollen. Transillumination was of no value. Application of suction was productive of a small amount of thick white mucus. The patient was treated with the usual local treatment of suction and nasal irrigations. An autogenous vaccine made from the nasal discharge was administered. No improvement occurred under one year's treatment, and a roentgenogram of the sinuses revealed that the frontal sinus was represented by only a bud in the ethmofrontal angle. Both antrums were hazy and the ethmoidal region was not as clear as normally. July 28th, 1923, the right antrum was punctured and the return specimen showed a flocculent, purulent discharge. The left contained less discharge. A bilateral intranasal antrotomy was done and the tubes allowed to remain in place for two days. On August 27th, patient had an irritable night from her asthma, and both antrums were irrigated. The return flow was only slightly stained. During the winter, the patient continued her nasal treatment and was free from asthma until April, 1924, when she contracted an acute cold. When the acute symptoms were subsiding, her asthma began to return. The antra were again irrigated, with relief, and the roentgenogram showed the proper sinuses to have developed from the previous bud to a level of the highest part of the orbital ring, and the general haziness of the antra and ethmoidal regions had disappeared. The child was on a regular diet during this winter.

Case 2. K. S., aged 9. First seen December 20th, 1923, at the Baptist hospital, where she was confined to her bed with an acute myocarditis and an acute nephritis. Examination showed the nasal membranes to be coated with a thin mucopurulent discharge, and the lymphoid tissue of the nasopharynx to be hypertrophied and red. The tonsils had been previously removed, but there was some reformation in the left fossa. The roentgenogram revealed a general cloudiness of the sinuses, with the right antrum markedly cloudy. The frontals were symmetric and had developed to the level of the orbital ring (slight retardation but not arrested as in Case 1). December 20th, 1923, the left tonsil fossa was cleansed of the reformed lymphoid tissue, a bilateral intranasal antrotomy was done, and the tubes were allowed to remain in place for one week, as the discharge was profuse. An autogenous vaccine was made, and on the 31st of December, 2 mm. of vaccine was administered. The reaction was very marked and produced a slight disturbance of her cardiac compensation. Local treatment and irrigations were continued. On March 15th, transillumination showed the left antrum to be darker than the right, but suction of the sinuses was negative, so the antra were not punctured and irrigated. On April 9th, both antrums illuminated equally and satisfactorily clear, and again the suction was negative. The patient's general condition had greatly improved, the heart action was reported as normal, and the urine was free of albumin and casts. The roentgenogram made September 9th showed the frontal sinuses to have grown at about the normal rate, and the ethmoids and antra were clearer than the previous plate. Inasmuch as the child did not have an arrest of development of her sinuses, there was no phenomenal growth of the sinuses under treatment, but only the normal progress.

Case 3. N. W., aged 11. Chief complaint, chronic nephritis, for which no cause could be found by the pediatrician. Examination showed the nasal membranes to be dirty and coated with a thin mucopurulent

discharge. The inferior and middle turbinates were very small and undeveloped. Transillumination of the sinuses was unsatisfactory, because both frontal regions failed to transilluminate and the cloudiness over the antra was equal. The roentgenogram showed an arrested development of the sinuses, the frontals being absent and represented by a small bud in the ethmofrontal angle. The right antrum was cloudy and the left frontal clear. There was a small piece of lymphoid tissue in the right tonsil fossa, but the left tonsil had been successfully removed. March 4th, 1924, the reformed tonsil tissue was removed and a bilateral intranasal antrotomy was done. The right antrum contained a thick, yellow discharge, but the left antrum was practically clear (confirmation of the roentgenogram findings). The patient remained in the hospital for one week, but the left tube was removed on the third day. The child's father was a dentist, and as they lived in a distant city, he was instructed in the irrigation of the antrum, which he continued for six months, as the urine would show albumin whenever the irrigations were discontinued. The roentgenogram taken on September 8th showed the arrested development to have remained the same, but the right antrum was only slightly more dense than the left. Both antrums washed clearly, but inasmuch as her sinuses had not begun to develop, and because of the persistency with which the nephritis reasserted itself, we advised continuation of the treatment.

Dr. Joseph C. Beck, Chicago: I had the pleasure of seeing these cases of Dr. Arbuckle and examining them in St. Louis, and I can vouch that they were splendid specimens of children.

There is one type of sinus disease in which absorption does not take place, where there is no systemic disease, but by continuity of structure a pneumonic or bronchitic condition exists, in which the sinuses are at fault.

Dr. Arbuckle spoke about unsatisfactory results from suction. I think, in some persons, when used not only by the physician, but by the patient at home before he presents at the office, it is a help. You can take a canula, reach into the nose and use suction directly, and thereby withdraw a considerable quantity of pus from under the middle turbinate. In that way it is quite satisfactory.

Dr. Millard F. Arbuckle, St. Louis, Mo. (closing): I want to say a few words about gas anesthesia. The gas anesthesia that I use in these cases is continuous gas-oxygen, and it can be used as long as desired. Of course, a postnasal plug is put in. I think there is less shock with gas anesthesia, and less damage to diseased kidneys or other organs, than with ether. We had difficulties at first in the administration of gas, but after a prolonged effort, it works better for me than anything else.

The cause of the edema, worked out by Doctors Marriott and Clawson, is a substance in the blood which is of bacterial origin. This substance apparently causes a change in cell tension throughout the body, setting free water into the intercellular spaces, where it remains. The sudden loss of edema after removal of large quantities of pus, in these cases, is further evidence of restoration of cell tension.

I have never used Dr. Shea's catheter, but I think it is a good scheme, and I hope to try it out.

BELL'S PALSY; FOUR CASES OF INFECTIOUS ORIGIN.

VIRGINIUS DABNEY, M. D., F.A.C.S.

WASHINGTON, D. C.

In the year 1823, Charles Bell, an Englishman of keen powers of observation, noticed that in cases of facial paralysis, whenever the patient tried to close the eyelid of the affected side, the eyeball would roll upward and, upon persistent effort, outward as well. Facial palsy, as it was then called, of unknown, or at least problematic origin, characterized by this sign, came to be called "Bell's Palsy". The French observers in particular were struck by this symptom, and due to Bell's persistence and apparent originality of perception, consistently refer to it as "the sign" or "phenomenon of Charles Bell". From the very beginning, cold was thought to be the cause of this paralysis of the brow, cheek, lip, eyelid, and, when involving that portion of the nerve lying within the Fallopian canal, taste and hearing as well. It is interesting in passing to recall the number and variety of diseases in which chilling of the surface of the body has been from antiquity regarded as causative of disease, whether it be pneumonia or chilblains, acute nephritis or coryza, diarrhea or lumbago. The prevalence of the infectious diseases during cold weather seems to those ignorant of disease processes (not all of them laymen, at that) further proof of this belief, wherein they fail to realize that warm weather brings with it the open window and door, the admission of fresh air and sunlight, to which no germ is long resistant.

In the year 1889, both Charcot, the neurologist, and Neumann, the otologist, spoke disparagingly of cold as a cause, and both seemed to feel that an inherited susceptibility to nervous disease was responsible for the paralysis. In support of his belief, Neumann gave a history of one family in which three members were afflicted with this disorder. Bell was able to produce experimentally all the classical symptoms by severance of the facial nerve, but, as in the beginning of his

observations, concerned himself chiefly with the eye phenomena. He regarded the rolling upward and outward of the eye as "physiologic", due to the exaggerated effort to bring into play the now paralyzed orbicularis palpebrarum.

It is, perhaps, almost essential to a proper understanding of the different phases of this paralysis, that the course of the nerve involved be clearly kept in mind. The most convenient and logical division for this purpose would seem to be into three sets of branches: 1. Those given off within the Fallopian canal; 2. Those extracranial, and given off before the terminal division; 3. The temperofacial and cervicofacial divisions. This threefold division is not merely an anatomic nicety, but will explain why some cases of Bell's paralysis show ear symptoms like tinnitus and slight deafness, indicating involvement of the chorda tympani and stapedius, while others are complicated by perversion of taste and impaired motility of the tongue, showing implication of the chorda tympani and glossopharyngeal. Again, there may be nothing but uncomplicated facial paralysis, where the inflammation attacks the nerve after its exit from the stylomastoid foramen.

Careful studies of many temporal bones seem to justify the conclusion that the right stylomastoid foramen is generally more constricted than the left, and that this difference is found more often in the female. Thus, these two facts are sometimes regarded as predisposing causes of Bell's palsy, inasmuch as women are certainly more often afflicted than men, and a narrow canal exit would give a pressure paralysis in the presence of an effusion. Again, clinically, this theory seems to be borne out by experience, as right sided affections are more often seen in this disease. It is my firm conviction that every case of Bell's paralysis will be found to be of toxic origin, if care is exercised in the search for such a cause. It bears in several respects a very striking resemblance to herpes zoster, not the least of which is the cause: infection. I have not seen this comparison drawn by any writer, though it is so suggestive that someone must have noticed it long before my time. Herpes was long supposed to be due to exposure to cold and to many other fantastic things, but we now know it to be result of infection of the ganglion. If exposure to a draft or cold wind causes facial paralysis, why doesn't it cause a simultaneous facial neuralgia, as the trifacial nerve is much more easily disturbed by such a cause, and is much

more vulnerable in the face and temple, by reason of the superficial nature of its distribution? Yet, it does not do so. With a heavy protective coat of bone, periosteum, muscle and skin, is it likely, or even possible, that cold would attack that part of the nerve lying within the Fallopian canal? Yet it would have to do so to cause taste and hearing perversions associated with facial palsy, since a neuritis of the nerve external to the stylomastoid foramen would not give any symptoms other than the paralysis. On the other hand, infection through the blood stream readily reaches any nerve anywhere, and the close confinement in a small, unyielding, bony canal to which the facial nerve is subjected, renders a paralysis certain with the slightest effusion into the nerve sheath, such as is caused inevitably by an infection of that nerve. It is interesting to see that some of the older writers have shown sufficient courage and independence to consider "rheumatism" as a cause, and quite suggestive, when we realize that cold was thought for many years as a cause, in turn, of this rheumatism. Thus, we gradually arrive at the real cause: infection, which is, of course, the cause of rheumatism. Thus, these gentlemen were not far away from discovering the real cause of facial palsy, though they were in blissful ignorance of their proximity. Of course, syphilis, tuberculosis, pressure by growths, parotitis, encephalitis and hysteria may severally cause a paralysis of the facial nerve, as well as the familiar otitis media in its various forms, but this is not the Bell's palsy of classical renown. Bell, himself, and his followers referred to a paralysis of unknown or problematic origin.

Most cases of this trouble show either tongue or ear, or both, symptoms, associated with the facial distortion; in other words, the neuritis is intracranial, where, as I have pointed out, cold cannot strike, but where infection can easily do so. On the other hand, exposure to cold where a patient is overheated can readily be the exciting cause of an acute exacerbation of a chronic trouble in the tonsils, teeth or nasal sinuses, and thus bring on a toxic neuritis of the facial nerve. In fact, I was able to demonstrate a definite causal relationship between each of these foci and a Bell's paralysis in the four cases which form the basis of this paper. Not only so in my own cases, but I go so far as to say that I have never seen a case where I was not able to find an infectious cause. To me

it seems the only reasonable explanation, and cold at best merely a contributory factor. I have been particularly struck with the invariable presence of the nervous diathesis in every case that I have seen or have had under my own care, though I cannot make myself accept Charcot's belief that this is actually a cause. However, it is a most interesting phase of the disease, and certainly exerts some influence on the prognosis and length of convalescence.

Case 1:M. This is the case of a highly nervous young man, much overworked, very conscientious in the discharge of his duties, and hard pressed to support his family. Here is the clear cut background of neurosis upon which Charcot lays, with much justification, such stress. In the course of my examination, I observed that he had bitten his nails down deeply, being merely another nervous sign. He had a complete paralysis, with all the tongue, facial and ear symptoms, of three days' duration when I first saw him. The onset was sudden, and attributed by him to exposure to a draft when he was overheated. He proved to have acute empyema of the left antrum, and acute exacerbation of a chronic tonsillitis. Irrigation of the antrum and swabbing of the tonsils with silver nitrat resulted in marked amelioration of all the paralytic symptoms in 36 hours, and complete disappearance of all of them in ten days. The antrum was certainly the source of the infection, as his tonsils were not removed and his paralysis disappeared. However, a tonsillectomy should be done, of course, to prevent possible reinfection, and he was so advised.

Case 2:B. Here the nervous element was not prominent, but was certainly present. It was that of a government clerk, whose trouble had been well established for four days before I saw him. The mouth and cheek on the affected side were immobile, but the eyelids merely slow in closing, and the tongue only partially affected, there being some difficulty in keeping food out of the hollow of the cheek. The ear was not involved. He had had another attack five years before, and attributed both attacks to exposure to cold wind while driving a car. He had buried tonsils with surface plaques of caseous material, and palpable glands at the angle of both jaws. I removed his tonsils under novocain-adrenalin infiltration, and in two days the eye was closed easily and normally, and in two weeks there were no signs of any paralysis.

Case 3:G. This man had had his second attack in six

months when I saw him, in consultation, and gave as the cause sitting on the ground when overheated from playing golf. I was naturally sceptical, holding the views of etiology that I did, but especially so as the day referred to was very hot and even the ground seemed warm. At this time, the cause seemed to me to be the tonsils. His paralysis was absolute, such as is seen only in severance of the nerve, with epiphora and all the other distressing symptoms. The time honored treatment of strychnin, hot and cold applications and faradism were all tried, and in two months most of the trouble was gone, though it was three months in all before the condition approached normal. Even then the eyelids looked sluggish. I was suspicious of his teeth, but contented myself with a recommendation of tonsillectomy. I lost sight of the case, not being mine, for a year, at which time he had his tonsils removed, not for the relief of the paralysis, but merely because he felt that his throat was uncomfortable in winter. Three months later a twitching of the mouth and eyelids, difficulty in closure of the eye and inability to whistle made him and his numerous advisors feel that my theory of infection was wrong, as the tonsils had been out three months. However, it in no wise vitiated my theory, as I pointed out an upper bicuspid, whose removal showed a large apical abscess and resulted in an apparently permanent cure of the paralytic signs. He still has some teeth that I think unsafe, and that may cause a recurrence. While outwardly not nervous in the laymen's view of the word, yet this patient is one of the repressed, nervous types whose highly organized nervous system is subject to an exaggeration of subjective symptoms.

Case 4:P. This case was seen after a week's duration of the paralysis and was most distressing to witness, as she was a pretty woman, very sensitive because of her disfigurement, who had had neurasthenia six months before. The eye signs were most pronounced, chiefly because she had little play of expression and the lips were not so apparent in their sluggish movement. However, her tongue gave her the most trouble as she often bit it while eating, and food accumulated in the cheek. She thought her trouble was due to washing her hair and allowing a draft of air to strike her head while wet. There appeared nothing wrong with the nose or throat, unless it was a slight redness of both tonsillar pillars, and the small tonsils themselves were free of exudate, but, noticing a

slight swelling over an upper molar I elicited the fact that she had had toothache for ten days. An X-ray picture showing some trouble at the root of that tooth, I urged immediate extraction, which was followed in 2 days by relief of the eye symptoms, and in ten days of the whole paralysis.

Conclusion.

To the objection that the disappearance of the facial paralysis and the removal of a focus of infection may be merely a coincidence, and that cases recover without any surgical intervention, the reply is that this disease is singularly intractable under old methods of treatment, and singularly amenable to treatment by removal of infection; under nonsurgical treatment requiring from one to six months for either cure or even lessening of the symptoms, and under surgical measures showing improvement frequently in two days and always in seven, with definite cure in twelve to eighteen days, depending upon the duration of the disease before removal of a focus of infection was attempted.

It is here pertinent to state that of those conditions in the eyegrounds showing exudate and effects of infection, 60% are due to apical tooth abscesses, 25% to diseased tonsils, about 10% to nasal sinus disease, and the remaining 5% to infection elsewhere in the body, divided among foci in the gall bladder, genitourinary tract and the intestinal tract.

So far as clinical evidence can be conclusive, two facts stand out clearly in these cases and in all I have ever seen; (1) the definite connection, or at least presence of infection in cases of Bell's paralysis; (2) the existence of the nervous diathesis. I believe that every case will show some focus of infection, whether it be in teeth, tonsils, nasal accessory sinus or some other place, and that such infection is the true cause of every case of what we have recognized since the time of Charles Bell as "Bell's Palsy."

DISCUSSION.

DR. L. E. BROWN, Akron, Ohio: At the outset, I recognize that this subject is one of a neurologic character, rather than a topic which belongs exclusively to our limited field of practice. It is to the neurologists that we must turn to ask questions about Bell's palsy. But the first lesson to be learned is, that the human subject must be studied from the physiologic and the pathologic viewpoints as a unit.

The neurologists, as late as ten years ago, recognized and taught that the incidence of this particular affection occurs more often in males than in females; that the common age is between twenty and forty; that it is more frequent in winter and in temperate climates; that the typical cases are due to exposure and to the socalled "rheumatic influences" with infection; that a neuropathic tendency predisposes to it; that often ordinary cases are due to microbic infections similar to those of "colds" and influenza; and that the most frequent type is the peripheral facial, or intranuclear, which comprises over 80 per cent of all cases. My authority for these statements is Dr. Charles L. Dana, and he so writes in his Textbook on Nervous Diseases published in 1915.

It is easy to recognize that the neuroanatomic basis does enter into consideration as an etiologic factor. Philip, of Bonn, (quoted in Dessert, in 1899) made a study of a series of skulls to determine the terminal diameters of the exit of the aqueductus fallopii. Invariably, the diameter on the right side was smaller than on the left. In another series, a study of clinical records was made, and in a predominating number of cases, the paralysis was found to be right sided. The anatomicopathologic explanation given was, that as the result of exposure of the right side of the face to a current of air, evaporation of the skin took place, with an accompanying superficial vasoconstriction and a simultaneous deep seated congestion. As a result, the neurolemma of the nerve trunk became swollen and impinged on the neuroaxial fibers, because of the nonyielding construction of the stylomastoid foramen.

The most striking portion of the paper to me is that dwelling on the treatment. Operative intervention in the acute stage of the disease may be regarded as quite radical. The results of the author are indeed brilliant and spectacular. I wish his series comprised a much larger number of cases. If operative treatment in the acute stage following focal infection produces relief of paralytic symptoms in two or three days, and cures the patient in ten days to two weeks, a most decided forward step has been taken. I am free to admit my own results have not been so gratifying to myself or to my patients.

It is to be remembered, on the other hand, that many cases of the peripheral type tend to a spontaneous recovery in about two or three weeks, and the prognosis is invariably good. However, in the vast majority of cases, cure or recovery takes place in from eight weeks to eight months under any or all forms of present treatment. Slight traces are sometimes left of permanently impaired function, and it should be ever remembered, that in cases which show involvement of the upper neurons, complete recovery seldom, if ever, occurs.

It is apparent to all who see cases of Bell's paralysis, that a disproportion exists between the untold number of individuals who harbor a

nidus of infection and the resultant syndrome of the caption of the essayist's paper.

If we agree in toto as to the etiologic factors set forth in the argument, and accept the causes to be infection, demonstrable or cryptogenetic, on a neuropathic basis, it seems to me that the last word has not been spoken.

This paper is modern, scientific, and quite tenable. The lesson taught is that of careful, painstaking and very thorough examination, bringing to our aid all the modern means of diagnosis in the way of instruments and laboratory equipment. But when I contemplate the great disproportion mentioned a moment since, I am compelled to conclude that some reason exists to explain the all but universal infection in the tooth apex, the crypts of the tonsil, the sinuses, the gall bladder, the gastrointestinal tract—in other words, the ubiquity of cause number one set forth in your hearing—even when the neuropathic constitution is added as a second predisposing cause, and the relatively small number of cases of Bell's Palsy that result when these two causes become operative. It is true, that answer may be made, that paralysis of the great motor nerve of the face is but one of the syndromes when the two causes mentioned are in force, or even when a contributing cause, such as cold, draught of air, dampness, or any other factor which tends to lower resistance is added. This we well know is no longer a question that calls for affirmative and negative defense. It is not debatable.

Indeed, it is not hard to believe that some other cause than the two set forth in the paper must exist. The bacteriologist will explain in clearer terms what he means when he speaks of immunity and resistance. He will doubtless tell us why one is soon well under an attack of virulent infection, and why another succumbs under the invasion of an attenuated type of the same organism.

This hypothesis of an unknown factor seems tenable and when it, too, becomes the known factor, not only neuritis of the symptomatology described by Charles Bell, but many other manifestations of infection will have become common knowledge.

Dr. W. W. Pearson, Des Moines, Iowa: We have all heard complaints of disturbance of taste and sensation in the distribution of the branches of the seventh nerve given off in the middle ear. I wonder if they are not the result of a slight involvement of the seventh nerve, in which the typical Bell's paralysis is not fully developed. This same complaint no doubt results from the direct influence of a suppurative process in the nose and throat, the nerve terminals being directly acted upon by the resulting toxins. The question of focal infections carries us so far afield that we soon begin to speculate.

Dr. Dabney's report cannot fail to impress us with the importance of a thorough search for the probable cause of these cases of Bell's paralysis. No doubt, in a certain number of these cases we will be able to demonstrate focal infections, but in others, search as we will, we will not succeed in finding the cause.

Two unusual cases of seventh nerve palsy have come under my observation within the past few months. One was that of a baby about eighteen months of age. It was not possible to demonstrate the cause

in this case, and a thorough examination by an internist revealed nothing. The possibility of encephalitis could not be excluded. The other case was that of a gentleman above 40 years of age, who for a few years had carried a high blood pressure. In this case, the paralysis was well marked in the entire distribution of the nerve. Teeth, tonsils and everything had been cleared up months before, with the hope of influencing the blood pressure, and he had been constantly under the observation of a careful internist, so that focal infection in this case could be fairly excluded. The neuritis apparently was due to a circulatory condition. Recovery took place in about two weeks.

My greatest difficulty is in securing thorough searching examinations in these cases. After eliminating all the possibilities so far as the nose and throat are concerned, a certain number of these cases remain in which we have failed to locate the trouble.

For a number of years, it has been my thought that toxins originating in the intestinal tract are responsible for a number of those cases which we classify as focal infection. In a conversation with Dr. Novy, of the University of Michigan, about fifteen years since, when I was interested particularly in discovering the cause of many cases of low grade inflammation within the eye, I expressed the hope that further investigation would aid us very materially in discovering the origin of this trouble in the disturbance of function along the alimentary tract. His reply, however, discouraged me. He said that there were so many vital functions along the tract which could not be reproduced in the laboratory, that the solution, to him, appeared to be far off.

Since Dr. Dabney's report, I am sure that we will all be more critical in our search for the cause in these cases.

A FURTHER STUDY OF THE TOXIC EFFECTS OF LOCAL ANESTHESIA.

EMIL MAYER, M. D.

NEW YORK CITY.

The great importance of local anesthesia in our special fields of medicine has induced your Program Committee to request a further review of the subject, together with such comments as may be advisable, adding reports of further fatalities, in the hope that there may be full discussion, that individual views may be freely expressed, and conclusions reached that will result in the saving of human life.

Too much credit cannot be given to the Therapeutic Research Committee of the Council on Pharmacy and Chemistry of the American Medical Association, who give so generously so much of their time to the scientific problems of the Association, for instigating this research. They have been most helpful in every way to our Committees, responding eagerly to any demands made upon them, advising, appreciating and encouraging at every step.

The Board of Trustees of the association have realized the importance of the investigation, have cheerfully endorsed it, granted the necessary funds for its completion, and expressed their gratification at its successful outcome. Their loyal support is deserving of the highest praise and gratitude of the profession.

The individual members of the various committees have cheerfully accepted any detail assigned to them, have loyally given the best that was in them, devoting many hours unselfishly to the success of the investigation without recompense, save that of the knowledge of a duty willingly performed, and have worked in unison for the common good. As Chairman of these Committees, I beg to extend to them my grateful appreciation and to assure them that I have been honored by association with them, and have been profoundly touched by their many kindnesses, endorsement and unanimous support.

No small share of our appreciation belongs to those members of our profession who, relying on our honor, have given us their confidence and have related with meticulous care the details of the tragic occurrences they were unhappily forced to witness.

At the outset, we were met with the fact that we were entering on a subject that had never before been successfully attempted. It became necessary to devise means for conducting the investigation, and to plan for its study from every point of view. Mere tabulation would have been futile. We had to ascertain if deaths occurred, the drugs responsible, the symptoms of poisoning, and this included measures used to resuscitate as well as any contributory factors, a study of any necropsies, animal experimentation undertaken when necessary, and finally, after careful study, the presentation of such recommendations as would tend to lessen the number of fatalities and result in saving human lives. Two reports were published by my former Committees.

The report of the general Committee was published as a special article in the Journal of the American Medical Association (March 15, 1923), and given ten pages of its valuable space. It has been widely abstracted, and commented upon editorially in many journals; many letters and personal expressions have been received, all of them of hearty approval. This report, preceded by an analysis by the chairman of the Therapeutic Research Committee, Professor Torald Sollmann, was submitted first to the members of his committee, approved and signed by each. They expressed their appreciation of the work. The keynote of that analysis was in the statement that "the committee believes that the observance of the concentrations recommended will reduce the risks materially, if not altogether. This does not question the right of the surgeon to depart from these concentrations. But he may see that in departing from them he is treading on dangerous ground."

Pharmacologic studies became a necessary integral part of this investigation, and these were conducted for us in the laboratory of the department on Pharmacology, under the direction of Dr. Hatcher. In the annual summary of work done in that department of the Cornell University, this entire report appears, showing the result of studies made in that institution and with its cooperation.

WHAT THE INVESTIGATION HAS ACCOMPLISHED.

We have established the fact that fatalities occur more frequently than the occasional reports in Medical Journals would seem to indicate; deaths ascribed to a local anesthetic are frequently due to entirely different causes, often one drug is held responsible where others far more toxic have been combined. We have presented a new nomenclature. The symptoms preceding death have been clearly elucidated, as well as the rapidity of their occurrence; we have therefore concluded that only such deaths as occur within a few minutes with the same symptoms—convulsions, paralysis of respiration and coma immediately after injection—are true anesthesia deaths. There seems to be an equal sensitiveness to these drugs in the throat, nose and urethra. The amount of epinephrin used has been carefully considered, and further studies of this drug have been recommended.

Injection of cocain in the mucosa or subcutaneously have been found to be very dangerous. The use of cocain paste has been condemned. (This has created some criticism as will be shown later on.) Certain specific instances, as the presence of trauma and constitutional diseases, are mentioned as adding to the danger.

Generally speaking, it was found that there was a tendency to use far greater concentrations than were recommended by the manufacturers or originators of local anesthetics, and each operator followed his own inclinations. No official statements as to the strength of these drugs had ever been made. This latter the Committee has attempted to supply.

We believe it to be indisputable, that local anesthesia may be induced safely by the methods recommended by the Committee. The committee does not maintain that anesthesia may not be induced in other ways with comparative safety, but it does maintain that death follows their use in the ways that it condemns.

Toxic effects have come on so suddenly, that a great variety of drugs have been used to resuscitate. Those likely to be of service have been mentioned.

While the reports have been most kindly received, there have been criticisms. The 1920 report was thus criticized:

"In the entire experience of all the members of the Committee of the American Medical Association to investigate local anesthesia in nose and throat work, there has not been

a fatality, yet they accept scattered reports of deaths from local anesthesia from men, probably of much less experience than themselves, in which there is no mention of a single autopsy having been performed, to eliminate the other causes of sudden death." To this I replied:

"While it is true that *all* of the three members of that Committee had no fatality, this was mentioned to indicate their freedom from bias in recording fatalities, and no inference could be drawn therefrom that it was their skill that prevented such an occurrence. The confidential nature of communications prevented the Committee from stating who had the unfortunate experience, but it should be stated unequivocally that death occurred often in the practice of men fully as prominent and as competent as any of the Committee, indeed they were known to us as especially capable men of large experience.

"As to the statement, not a single autopsy, attention is called to the fact that this is untrue. That report mentioned *three autopsies*. Cases 3A, 3B and number 10."

Criticism of this sort involving gross misstatements are vigorously resented. The critic was informed by me of the facts. He has had neither the grace nor the courage to retract his statements.

Cocain Paste.

The recommendations as to the use of cocain paste have occasioned protest by some of the small minority who favor the procedure, notably among our confreres in England. Their statements are herewith appended:

Among the many Journals reviewing the final report was the London Lancet, May 17, 1924, page 871.

After presenting the recommendations of the Committee editorially, the Lancet states that "the present report will have done a very useful service if it results in the fixing of recognized limits for the concentrations and doses in which local anesthesia should be employed." (The following communications were published in the same journal on May 31, June 7 and June 14th.)

Lancet, May 31, 1924.

"Sir:—Among the recommendations of a committee of the American Medical Association (see the Lancet, May 17th, p. 1013) is the injunction 'that cocain paste should never be used,' presumably because of its toxic effects. I do not

know what exactly the committee means by 'cocain paste,' but if it is the mixture that goes by the name in London, then I, for one, am at a loss to understand the downright condemnation. The paste, as we use it, consists of pure cocain hydrochlorid powder mixed with a solution of adrenalin (1-1000) to the consistence of a mud or paste, and I have been employing it regularly, five or six times a week, for the last ten years as my mainstay in anesthetising the nose, during the whole of which period I have not had one single case of cocain poisoning, not even of the milder variety. This claim may seem surprising, and the surprise will not be lessened, when I say that in the septum resection operation, I habitually use as much as from 4 to 6 grains of cocain for each case. Not only so, but when a general anesthetic is being used for that operation, I apply the paste to the septum as a preliminary, and my anesthetist friends can testify that that step is followed by no evils.

"The secret of safety seems to lie in the judicious use of adrenalin. In making his application, let the surgeon paint the mucosa first of all with pure adrenalin solution alone and undiluted, in order to induce the local anemia that minimizes absorption, and, at the same time, denudes and exposes the sensory nerve endings. Then the cocain-adrenalin paste may be safely applied. It may, I think, be laid down as a rule, that cocain, whether in powder or in solution, should never be employed without adrenalin. The paste is, we must remember, not suitable for all purposes. I have never ventured to use it in the pharynx for example.

"I am, Sir, yours faithfully,
"Dan McKenzie."

The following week, this letter was published:

Lancet, June 7, 1924.

"Sir:—Dr. Dan McKenzie deserves our thanks for setting forth his method of using pure cocain hydrochlorid powder mixed with sufficient adrenalin solution to form a mud or paste for direct application to the nasal mucosa for producing local anesthesia. Further, that thus applied, he habitually uses as much as 4 to 6 grains of cocain for each case, and never has he had one single case of cocain poisoning. Assuming that this is the cocain paste referred to in the report of the Committee of the American Medical Association, which they recommend should never be used, Dr. McKenzie's

letter is a timely and authoritative protest against such sweeping general statements.

"Yet it seems curious to find, that Dr. McKenzie goes on to lay down a rule that cocain, whether in powder or in solution, should never be employed without adrenalin, and the need of caution in the exhibition of all local anesthetics must be borne in mind, nevertheless, many of Dr. McKenzie's friends regard it as one of the unrivalled advantages of cocain over all other local surface anesthetics, that cocain may be used for light anesthesia for the nasal mucosa without the disadvantages occuring from the application of the adrenalin. Probably, Dr. McKenzie referred to the use of strong application for pronounced degrees of anesthesia, but if this be so, it only emphasizes the need of care in avoiding such generalizations as Dr. McKenzie himself condemns, which tend to hinder rather than help us in arriving at sound conclusions.

"Patrick Watson Williams,
"Clifton, Bristol."

This was replied to in the Lancet June 14, 1924.

"Sir:—If Dr. Watson Williams will turn again to my letter, he will see that my reference to the advisability of always combining adrenalin with cocain is preceded by the words, 'It may, I think,' whereby I intended to convey the impression that it was a suggestion I was making rather than a rigid generalization, the perils of which I am aware of. For the point is, I know, one for debate. No one will, perhaps, quarrel with the dictum that, as a rule, and particularly in extensive anesthetizations, adrenalin should always accompany cocain. In the matter of light anesthesia of the nose, however, as for examination purposes, the question is, as Dr. Watson Williams quite correctly implies, not by any means so simple. In light sprays, adrenalin frequently sets up an annoying reaction, with sneezing and running at the nose, which I have known to last as long as forty-eight hours, and to be a cause of much bad language, directed, and with perfect justice, to the rhinologist's address. But on the other hand, I have occasionally seen cocain syncope follow even a trivial spraying of the nose with pure cocain solution; the drug is then so rapidly and completely absorbed.

"So there the problem stands, and each man must solve it for himself. Personally, I prefer a swearing patient to the

fainting patient. But I am quite ready to admit that others may cherish the opposite opinion. May I add that syringing the nose with warm saline solution after adrenalin-cocain spraying can, as a rule, but not always, be relied upon to avoid any subsequent irritation? It is curious that so violent a reaction seldom follows the more continued anesthetic applications needed for operation.

"Dan McKenzie."

This criticism is quite different from the previous one. It contains a statement of the individual experience of men of undoubted ability, and expresses their firm convictions. There can be no quarrel here. They are entitled to their opinions while we reserve for ourselves the same privilege.

We were led to our conclusions by our knowledge of deaths reported to us following the use of cocain paste; one undoubted death in the last series, two others occurring previous to the time specified in our investigations. As the sole contention of the *small minority* who advocate the use of this concentration was that it produced such profound local anesthesia that there could be no absorption, and hence it was far safer than weak solutions, even absolutely safe, the bona fide deaths disproved their contention. Hence our decision.

We are not always in accord with our English confreres as regards therapy. We do not understand why Dr. McKenzie ever uses profound local and general anesthesia at the same time. This seems to be quite the custom in Britain, for I note (British Medical Journal, June 21, 1924, p. 1091) a report of the meeting of Scottish anesthetists, which mentions "the use of local anesthesia after O'Malley's method and light chloroform with it." It assuredly is not the custom with us to use both forms of anesthesia at one time, and the experiments of Dr. Macht, reported by me in the Journal of March 15th, states that the combination adds to the danger.

Our English confreres use chloroform very freely. We do not.

This subject, far from being controversial, must submit to the test of time. It is very interesting to note that Dr. McKenzie has seen serious toxic effects from small doses of cocain, and never from the high concentration. Time alone will show which is right. In the meantime speaking for my committee, we do not retract our condemnation of this dosage.

Substitution.

The too frequent mistakes of substitution of one drug for another has been emphasized, and suggestions made toward their prevention.

Unmeasured Quantities of Solutions.

We have noted that unmeasured quantities of solutions, notably cocain, are used. A cotton tipped applicator is dipped into the bottle containing the solution and then applied to the parts. When asked how much they used, they do not know. If the solution would be placed in a graduated measure, the resultant liquid after the swabbing would enable the operator to say at least that not more than so many minims were used. The same argument applies to the spray. *Always measure the quantity of any solution used.*

In order to secure further confidential reports of recent fatalities for further study, and to answer any queries of members of the profession who may seek information on this subject, the Chairman of the Therapeutic Research Committee appointed a permanent Committee on toxic effects of local anesthesia, to serve at no cost to the Association. Dr. Elliott C. Cutler, Lakeside Hospital, Cleveland, Dr. Robert A. Hatcher, Cornell University Medical College and Dr. Emil Mayer of New York have agreed to serve under the Chairmanship of the latter.

The Permanent Committee.

Certain toxic effects and fatalities have come to our notice since the report was prepared, and these are here presented. It may be stated that one fatality was published, while the others came direct to me as chairman of the permanent committee. We have every reason to believe that, by our publication, we have stimulated the profession toward more frequent publication of accidents occurring in their practice. One writes, "my conscience will not permit me to withhold from you the history of the following fatality."

Among the communications received was the following: "I note your recommendation that 1% procain is advised. How do you reconcile this with the fact that dentists use 2% solutions?"

To this I replied that the opinion of the committee, and that of very many men with whom we conferred, was that 1% procain solution produced complete anesthesia; that one

man of large experience invariably uses ½% solutions to the complete satisfaction of his patients and himself, and that I could not accept the dictum of dentists until they had shown by experiments that 1% was insufficient where 2% was successful.

Additional Reports of Toxic Effects Following Local Anesthesia.

These have been received since January 1, 1924. These are in reference to:

A. *Adrenalin.* 1. I was called hastily to see a patient who was dying from the effects of adrenalin administered by mistake.

2. I know of one death from that drug.

B. *Cocain*: 3. Alarming symptoms occasioning great anxiety followed after packing the nose (both cavities) with 10% solution of cocain.

C. *Butyn*: 4. I use 8 c.c. of a 2% solution of butyn for injection in tonsillectomies; out of 300 cases there were four cases of severe convulsions followed by profound shock.

(The manufacturers of butyn state distinctly that one per cent only of that drug should be used for injection.)

(One is compelled to conclude that the writer has a special providence watching over him. E. M.)

5. I injected 12 c.c. of a 2% solution of butyn for tonsillectomy. Violent convulsions and coma followed. The patient recovered!

These last cases bear out the contention of the Committee that the tendency in the profession is toward excessive doses of these drugs.

6. We have been using butyn in our local work for the past two years, and have never noticed any toxic effects from the use in the strengths in which we use it. Our practice is to paint the throat—the pillars—with 10% butyn twice, at intervals of 5 minutes, and then, just before operating, inject about the tonsils with 1/10 of 1% butyn solution with 1 drop of adrenalin to each dram of the butyn solution used, in all, about 6 drams of the solution for the two tonsils.

We have not tried butyn in the eye. We have tried it in the nose for submucous work, using 5% solution with adrenalin the first packing, repacking at the end of 20 minutes with 5% butyn without the adrenalin. We find that when used in the nose, the anesthesia is wonderful, but the postoperative

edema is so severe and lasts so long, that we promptly discontinued the use of butyn for intranasal work.

Six additional fatalities following the use of local anesthetics have come to the notice of the chairman of the permanent committee. These came too late for inclusion in the report of March 15th. None has come since publication. One of these was published in the Laryngoscope (November 15, 1923); the five remaining were reported voluntarily to the Committee.

Two additional ones were received, but were manifestly not due to the drug in question, and they were excluded from the list. One followed an injection of novocain for tooth extraction. Death in the chair without convulsions. Autopsy showed grave valvular lesions of the heart.

The other was alleged to be due to butyn. Death occurred four days after an operation, with all the symptoms of septic infection. Aside from the sepsis, we feel that death must follow promptly with all the symptoms, to be acceptable as a death from poisoning. These two were rejected. The remaining six were from:

Procain 1
Cocain 1
Cocain-Procain 4

Two were for nose operations, and four tonsil operations. Four were males, two females.

Cocain-Procain Death. As in our former report, physicians state that death was due to procain. Careful investigation shows, in nearly every case, that cocain was used with it, and hence both drugs must be mentioned as causal factors in the poisoning.

Here is a typical case:

Female, age 17, came to clinic for tonsillectomy. Patient sitting. *Throat was swabbed a few times with a 10% solution of cocain* (amount not stated). About 2 c.c. of a 1% solution of novocain were injected; no adrenalin was used. Sudden cyanosis, fell forward unconscious; artificial respiration, pulmotor and stimulants to no avail. Life pronounced extinct after four hours. Necropsy: Edema and congestion of lungs and brain. No lymphoid hypertrophy.

This was reported as a novocain death. It is manifestly not a death from that drug, nor can we believe that 2 c.c. of a 1% solution of novocain could cause death. Another death

is recorded as due to procain that seems to me to be doubtful. Here are the facts:

Male, age 39. Had hypertension and nephritis but considered a safe risk. Tonsils badly infected. Tonsillectomy. Patient received hypodermic of morphin and atropin (amount not stated) ½ hour before operation. Semireclining position. 0.5% solution of novocain containing 4 drops of 1-1000 adrenalin solution to the ounce. 1 drachm injected in each tonsil. Both tonsils injected before beginning operation. Right tonsil removed without any difficulty. As the left tonsil was being removed, patient suddenly had convulsion. Became cyanotic with dilatation of pupils and cessation of respiration. Artificial respiration. Patient began having shallow respirations lasting about five minutes, remaining cyanotic. Death soon followed. No autopsy.

An individual with chronic nephritis and high blood pressure has succumbed after a small dose of a very weak solution of novocain. In the opinion of the writer the drug may only be considered as a contributing cause at the most.

Summary.

It is interesting to note the ages of these patients; they were, 14-17-20-22-34-39 respectively, an average of 24½ years, indicating a susceptibility of the young.

A note of warning against the combination of cocain with other local anesthetics, except in the smallest doses of the former, must be sounded. At the risk of reiteration, the method of using unmeasured quantities of cocain solution should be discontinued. It is unscientific as well as unfair to our patients. The use of a minim measure will enable us at least to say that of a certain number of minims there remained so many, consequently not more than so many minims were used, and the amount can then be fairly approximated. That certain people have an idiosyncracy to cocain is no doubt true.

We are in hopes that a continued study of these fatalities will throw some further light on this question, and have agreed to serve on the permanent committee at our own expense, in the hope of receiving further reports, and that the profession will cooperate by publishing the facts or informing us of any deaths they hear of.

All communications will be considered confidential.

We are grateful for the many kindly words of approval and sincerely trust that by our labors we may prevent the waste of human life. We have demonstrated that it is possible to secure information as to medical matters by certain methods, and that conclusions may be reached by following carefully formulated plans as in an engineering problem. In this instance, we believe that we have demonstrated that it is possible for the physician without laboratory training to present a scientific study of a given subject, offering a constructive report with such recommendations as will render safer and more effective one of the most valuable and important aids to our therapy.

DISCUSSION.

DR. A. E. BULSON, JR., Fort Wayne, Indiana: The medical profession is indebted to Doctor Mayer and his Committee for the valuable work accomplished, and the deductions drawn from their investigations. The plans followed were without objectionable features, and had the sole aim in view of arriving at trustworthy conclusions concerning the toxicity of the commonly used local anesthetics, and the formulating of rules covering safety as well as efficiency in their use. The recommendations of the Committee deserve endorsement by this great body of specialists, each and every member of which uses local anesthetics, and, therefore, is interested in safe procedures. The objections and criticisms that have been offered by a limited number of men are not worthy of serious consideration in the light of the findings of the Committee. Because some experienced and competent surgeons have not had unfortunate results, must not be used as a criterion when other equally as experienced and competent operators have been unfortunate.

The trouble has been, that until this Committee began its work, we did not know of some of the dangers of local anesthesia as practiced by a considerable number of the medical men, for it is a lamentable fact that doctors seldom record their failures, especially when it concerns the life of the patient, and even now the Committee is reasonably certain that they have but scratched the surface, so to speak, in bringing to light serious toxic symptoms and even fatalities that would make their argument stronger.

In dealing with the subject of the use of local anesthetics, well may we ask, "Why play with fire?" A large number of those having large clinical experience offer testimony to substantiate the contention of the Committee, that entirely satisfactory anesthesia may be obtained by adhering strictly to the rules recommended by the Committee as being within the range of safety, so why persist in following a practice that had been proved to be dangerous? In fact, in the face of the recommendations of this Committee, which has studied the subject exhaustively and brought forth evidence to sustain its convictions, is it not dangerous from a legal as well as professional standpoint to disregard the recommendations that have been made? The use of amounts and concentrations greater than those recommended by the Committee, and shown to

be not only unnecessary but dangerous, must of necessity reflect unfavorably upon the operator who may be unfortunate enough to contribute unwittingly to a tragedy. To use cocain paste or concentrated solutions of any of the local anesthetics in the manner described and recommended in some of the older textbooks, is to invite dangerous symptoms or even death of the patient, for we now know that such unfortunate results have occurred, and those following the practice who have not come to grief should bow down to an all wise Creator who has protected both patient and operator. Furthermore, we must not be deceived by the specious plea that cocain paste or concentrated solutions of any of the local anesthetics lose their possibility of harm in the presence of epinephrin, for it has been shown, and this Committee whose report we are discussing later will show, that epinephrin is not without danger when used on some patients; and the theory has been advanced, that a local anesthetic which exhibits toxic symptoms, by depression or paralysis of the respiratory centers, will show a greater tendency in that direction when epinephrin is employed in conjunction with the anesthetic.

Finally, is there one logical reason why we should not endorse the work of this committee and adopt the recommendtions of the Committee, when they offer not only reasonable safety but efficiency? I, therefore, move that this organization formally approve the recommendations of this Committee.

Dr. J. J. King, New York City: I wish to endorse and second what Doctor Bulson has said. This committee, appointed by the American Medical Association, has done probably the most notable work in laryngology that has been done in fifty years, and I am repeating and voicing the sentiments of the New York Academy of Medicine, where we are so familiar with Dr. Mayer's work, when I say this.

Instead of discussing the paper, I wish to recommend the report for your study and consideration. Let these reports be the gospel to guide you in your work in local anesthesia. They are remarkable reports. They are guides for safety and efficiency in local anesthesia. I have no doubt that, basing future work in local anesthesia upon these reports, we are standing upon the threshold of a great development along that line. However wonderful local anesthesia may be now, following these reports we are bound to do better work in the future.

Dr. Otis R. Wolfe, Marshalltown, Iowa: At the request of the committee, I wish to report the following case:

A young housewife, of about thirty, sent to the hospital for a submucous section to correct obstruction to breathing. General health apparently good; history as given, negative. At 7:30 the next morning, she was given morphin 1/6, scopolamin, 1/150, and taken to the operating room at 8:00 o'clock on the cart. She was placed in the semiresting position on the operating table, and two applications of cocain paste made to each side of the nose. Not more than three or four grains of cocain was used. Cotton soaked with adrenalin was placed in apposition to the mucous membrane, at the site of the intended incision. I expected to wait ten minutes and then begin my operation. In about six or seven minutes, the patient began to complain of shortness of breath, and said, "My heart is throbbing violently." Her head was lowered immediately and syrup of ammonia aromatic given. The pa-

tient's condition grew rapidly worse, skin waxy white and pupils widely dilated; pulse rapid—130, later 150, then could not be counted, later could not be palpated. This was not an ordinary fainting spell, and my partner, Dr. Walker, and myself both felt that the patient's condition was alarming. At this stage, 1½ ampules of obstetric pituitrin was given hypodermically; ½ ampule had been given two or three minutes previously. The pulse began to improve immediately, and the peripheral circulation returned. Operation was deferred, the patient was returned to her room, and she made an uneventful recovery expect for severe headache.

Through inadvertence, I was only then informed that albumin had been found in the urine. I subsequently learned that the patient had had several attacks diagnosed as "mild nephritis" during her pregnancy. Had I been informed of this condition of her kidneys, I would have hesitated before operating.

I will state that all our cases have a urine analysis routinely. I present this experience in the hope that I may get an expression of opinion, in the discussion, as to whether the symptoms that arose after the use of the local anesthetic were not brought about by the presence of the kidney condition.

Dr. Edwin McGinnis, Chicago: I had one fatality from local anesthesia during a tonsillectomy, and this case was reported to Dr. Mayer's committee. It is obvious from the report of this committee, that if we use local anesthesia, we must be on our guard continuously. The only reason I am here, is to bring before you the fact that you can do most of the operations on the nose, submucous resections and operations on the sinuses, with a minimal amount of cocain anesthesia. Nearly all the time, I am able to do a submucous resection with 10 minims of 20 per cent cocain solution, with comfort to the patient and satisfaction to myself.

Another point I want to bring out is, that for six years I have not mixed adrenalin chlorid solution with cocain solution. Some experimental work by a member of the Chicago Laryngological Society brought out the fact, that if you combine adrenalin and cocain, or procain, you increase the intracerebral venous pressure. It is far better to make an application of adrenalin, and then wait a few minutes to apply the local anesthesia.

Dr. H. V. Dutrow, Dayton, Ohio: This Academy and the profession at large owe a debt of gratitude to this committee for its great work in presenting, in a practical and concise way, the many dangers that attend the use of local anesthetics, and how to avoid them.

In studying the committee's report, one is struck immediately with two outstanding facts in the majority of the fatal case reports:

First: The number or duplication of drugs used.

Second: The strength of the drug or drugs used, especially cocain.

We know that all anesthetic preparations are more or less synergistic. We may know from experience the action of a single drug, but we do not know what it will do when used in combination with similar drugs. I, therefore, wish to emphasize the strict adherence to the use of one drug at a time in a given case.

The strength of the solutions used, especially cocain, as revealed by a study of the case reports, ranged from 4 to over 20 per cent. These stronger solutions were applied to the walls of the pharynx, in addition to the injection of a solution of novocain into and around the tonsil. The mucous membrane of the pharynx is very vascular and absorption is rapid; in addition the patient not infrequently swallows some of the solution, producing an additional toxicity through the stomach. Cocain paste, in my experience, has been most satisfactory. I have used it, exclusively, in all kinds of intranasal surgery for the past ten years, with excellent results.

Many of us can recall case after case of cocain poisoning only a few years ago, when it was common practice to pack a nasal passage, without any preliminary ischemia, with pledgets of cotton or gauze saturated in a four per cent solution of cocain, carefully placed in full contact with the vascular inferior turbinate and allowed to remain for from one-half to one hour for complete anesthesia.

Cocain paste must not be used in any case without first shrinking up the nasal mucosa with 1/1000 solution of adrenalin. It is local anesthesia that we desire, not blood stream toxemia, hence the necessity of reducing to the minimum the degree of absorption.

The adrenalin should be applied fifteen or twenty minutes before the cocain. The anesthetic is then placed only where you wish to operate, or over the nerve trunk that supplies the field of operation.

Dr. George F. Keiper, Lafayette, Indiana: It is a very unfortunate thing that the name "novocain" was changed to procain, because there is liability of mistakes in the use. "Procain" sounds too much like "Cocain." Cocain comes to us in three forms—large crystals, small crystals and flakes. The most toxic of these are the small crystals, next the large crystals, and least of all the flakes. Why that is, I do not know, but it is so. I notice Dr. Mayer used the word "powder" in describing the cocain mud or paste. I wonder if he means flakes. In making a cocain paste or mud, do not use cocain powder, but use flakes, reducing the quantity to the minimum. It is hard to obtain the flakes nowadays, because of the fact that addicts use these flakes, which they call "snow," and we have been at a great disadvantage in obtaining cocain in that form, the safest form in which it can be used for cocain mud or paste.

Dr. Harry L. Pollock, Chicago: I want to call your attention to a case that happened in one of our large hospitals in Chicago. I do not know the details of the first case, but the case was injected with 1 per cent novocain, and immediately afterwards died. The following day, a tonsillectomy was to be performed, and the same solution was used, a very small amount, and the patient went into convulsions and then coma, but finally recovered. The pharmacologist took this solution and examined it, and found it was not 1 per cent, as originally made, but from repeated sterilization and evaporation had become a 10 per cent solution. I give this as a word of warning. In our large hospitals, they mix up quantities and resterilize it day after day, and that causes a more concentrated solution.

Dr. Emil Mayer, New York City (closing): I am especially interested in the case presented by Dr. Wolfe, because it is another link showing that alarming symptoms do occur following the use of con-

centrated solutions, for there is no doubt that the case presented by Dr. Wolfe was one of cocain poisoning.

The statement has been made by our committees, that a minimum amount of cocain should be used. The whole thing, in my opinion, hinges on the fact that nobody, until this investigation was undertaken, ever took the trouble to investigate and find out what was the proper dose. Each one remembered the amount he was told to use when he first began, and followed that, without a thought of possible danger. But since the publication of these reports, I have had men come to me and tell me that they can get as good anesthesia in bronchoscopy with 5 per cent as they formerly got with 20 per cent solution of cocain. You know, in your own training, that you have been told in the matter of therapy to use any drug in the strength that will produce a physiologic effect, and no more. You can put your patient to sleep with one-quarter grain of morphin injected hypodermatically. Why give him a half grain? Why put him in danger of his life? Why use any of these drugs in dangerous doses? I have had men come to me and say, "Doctor Mayer, you have put the fear of God into me. I have learned that I can get the same results with smaller doses."

The case presented by Dr. Pollock is another instance showing that unless that pharmacologic investigation had been made, one would always have had the impression that the death was due to 1 per cent novocain, whereas it was 10 per cent. I say openly and publicly to you, that I do not believe you can kill a patient with one-half of 1 per cent solution of novocain used alone, unless you enter a vein.

One thing more, and that is that I have had some further communication with Dr. Dan Mackenzie, and in his final letter he says: "I admit the presence of ironic fate in medicine. We have had many instances where a course of treatment was beneficial with one man but proved tragical in another, so I am daily expecting, now that I have committed my views to the world in print, a fatality, from lack of caution. You have, you see, succeeded in rendering me uncomfortable, and all I have to console me is the hope that my plan of beginning the anesthesia with undiluted adrenalin will be sufficient to preserve me from disaster."

In conclusion I wish to say this, that while I have had my hand in many a brew in the many years (47) I have practiced medicine, I do not believe I have ever accomplished anything that I honestly think will be of more benefit to the profession and to the specialty that we honor ourselves by practicing, than in the careful and conscientious study during the past five years of the toxic effects following the use of local anesthetics.

BRONCHOPNEUMONIA AND ABSCESS SECONDARY TO TONSILLECTOMY.

JUDSON DALAND, M. D.†

PHILADELPHIA.

Death from bronchopneumonia following tonsillectomy is fortunately rare, as shown by Martin[1], who reports three in 14,690 operations.

The surgical risk in the first two cases reported was obviously great, and this clinical study was made with the hope of learning how to lessen this risk. For the sake of brevity, only important and positive findings are recorded.

CASE 1. October 8th, 1917, male, age 33, married. Family history negative. Recurrent furunculosis and a cardiac murmur since childhood. The murmur may have been due to hemorrhagic chickenpox or la grippe. Syphilis was diagnosed in January, 1917, mercury and neosalvarsan administered, and the Wassermann reaction was weakly positive on October 9th, 1917. He complained of nervous exhaustion, insufficient sleep, frequent violent headaches, causing intense suffering, and recurring severe epistaxis, in one attack losing approximately one quart of blood.

Physical examination showed moderate emaciation, hypertrophy and dilatation of the heart, aortic and mitral regurgitation, Corrigan's pulse, dilatation of the aortic arch and arteriosclerosis. Cardiac compensation was well maintained. The blood coagulation time was normal; erythrocytes 3,710,000 or 74%; hemoglobin 62%; leucocytes 5,400; differential leucocyte count was normal. The systolic pressure in the sitting position was 140, diastolic pressure 40, pulse pressure 100. The blood pressure remained unchanged in recumbency and, after ten bending movements, the systolic pressure increased 5 mm. Urinalyses were negative.

Dr. George Fetterolf found the tonsils large, chronically congested, and the seat of a chronic septic process. In con-

†Professor of Medicine, Graduate School of Medicine, University of Pennsylvania.

sultation, we decided to recommend the removal of the tonsils, despite the large mortality risk, due to a tendency to hemorrhage, cardiovascular disease, syphilis and furunculosis. This decision was also influenced by the frequent, uncontrollable and intensely severe headaches. These facts were placed before the family and, with their consent, the operation was performed by Dr. George Fetterolf, on October 15th, 1917, whose faithful, conscientious and skillful care of the patient commanded my admiration, and whose observations have already been published.[2]

The removal of the right tonsil, under ether, was followed by hemorrhage, lasting more than three hours, and it was estimated that four to six pints of blood were lost. The erythrocytes decreased to 2,850,000 or 57%, hemoglobin 58%. The leucocytes increased to 11,600, chiefly of the polynuclear variety. Later, the leucocytes increased to 12,400, and, finally, to 18,000 per cm. Before the operation, a statement was made that as the walls of the arteries were diseased, hemorrhage was to be expected, but it was not anticipated that it would be so difficult to control. After the operation, it was suspected that a pulmonary abscess would develop, in consequence of trauma and prolonged anesthesia, necessitated by efforts to control hemorrhage.

On the third day after the operation, the temperature was 100° F., and the improvement was sufficient to permit him to return home. On the fourth day, the leucocytes increased to 11,000, and the erythrocytes decreased to 2,880,000 or 53%, and the hemoglobin 58%. On the sixth day, the leucocytes were 13,000, the temperature was septic in type and later reached the maximum of 104.6° F.

The cough, which at first was moderate and dry, later became severe and paroxysmal, and accompanied by mucopurulent expectoration. Occasionally, the odor of the sputum and breath was putrescent, probably from the sloughing exudate in the tonsillar region but, later, when the throat healed, the putrescence increased, and four to eight ounces of sputum were expectorated daily, which was highly malodorous and contained many streptococci and capsulated diplococci, but no tubercle bacilli. On or about the seventh day, the physical signs of bronchopneumonia slowly developed in the right margin of the lower lobe of the left lung, and gradually extended, and later an abscess formed. Aspiration

of the abscess was advised and performed, with negative results. The patient steadily grew worse, and pulmonary edema caused rapid death forty-two days after the operation.

The autopsy showed pleural adhesions and a moderate sized irregular shaped abscess cavity, about the size of an egg, filled with pus, occupying the lower lobe of the left lung. The lung surrounding the abscess was pneumonic. The aortic leaflets were fibrotic and markedly retracted.

REMARKS.

This death was directly due to hemorrhage, secondary to syphilitic endarteritis, causing shock and increased susceptibility to the streptococcus in the infected tonsils, the absorption and inspiration of which was favored by prolonged etherization and trauma, necessitated by efforts to control hemorrhage. These conditions also favored embolism. In a similar case, even though the blood coagulation time is normal, the intravenous use of 10 c.c. of a 10% solution of calcium chlorid in redistilled water, daily for three days prior to a tonsillectomy, is recommended.

CASE II. January 24th, 1921, a spinster, aged 71, complained of constant and severe occipital headache, dyspnea on moderate exertion, occasionally awakening her from sleep, tremors of the hands and feet, which began two and a half years ago, constipation, occasional severe shooting pains across the lower precordial region, general weakness, nervousness and distress. She had infantile paralysis at the age of two, severe nervous breakdown at the ages of 39 and 70, and six months ago, slight apoplexy or thrombosis. Her suffering was such that she said death was preferable.

Examination revealed well marked senility, defective memory and power of concentrating, paroxysmal attacks of cough, aggravated by exertion, moderate obesity, chronic myocarditis and degeneration, cardiac decompensation, rhythmic clonic tremors of the hands and feet, extreme weakness, edentulous, and exaggerated knee jerks.

Erythrocytes 4,140,000 or 82%, hemoglobin 80%, leucocytes 5,600, polynuclear cells 57%, large lymphocytes 9.5%, small lymphocytes 33.5%. The anemia, leucopenia and lymphocytosis was believed to be due to the absorption of the toxins or microorganisms, or both, from a focus of infection.

In the sitting position, pulse 92, systolic pressure 170, diastolic pressure 110, which was unchanged by recumbency.

Exercise increased the pulse to 116, and caused marked dyspnea. Excepting for large amounts of indican and skatol, the urine was negative.

Dr. J. Leslie Davis[3], who has referred to this case in a recent article, reported the tonsils as of moderate size, and markedly septic and, in consultation, we agreed that as no improvement could be expected from medical treatment, so long as infection continued, a tonsillectomy should be recommended, despite the great operative risk, due to senility, adynamia, obesity, chronic myocarditis and decompensation, provided the brother of the patient approved after the gravity of the operation had been fully explained.

The operation was performed by Dr. J. Leslie Davis on February 6th, 1922, under local anesthesia, the patient having previously received, for several days, full doses of the infusion of digitalis and the tinctur of nux vomica. Owing to the condition of the tonsils and the weakness of the patient, the operation was prolonged, and was followed by considerable shock. During the night following the operation, pulmonary edema and congestion occurred, requiring active stimulation and venesection. On the third day, pneumonia developed in the base of the left lung. On the fifth day, both bases were congested and the entire lower lobe of the left lung was consolidated. Temperature was 104.6° F., respiration 60, pulse 130, cyanosis, delirium, albuminuria, hyalin and granular tube casts, and leucocytosis. Death occurred six days after the operation, before a pulmonary abscess developed.

Remarks: In similar cases, in the future, the complication of pneumonia or pulmonary abscess may be diminished, if antibody resistance is increased by the preoperative use of an autogenous vaccine, and by devising a technic by which the duration of the operation and anesthesia is reduced to a minimum, thus lessening shock and the risk of embolism or the absorption or aspiration of pathogenic microorganisms.

Case III. December 11th, 1922, male, age 61, first seen in 1904, at which time he was suffering from neurasthenia, intestinal toxemia and partial cardiac decompensation, secondary to chronic valvulitis. The family history revealed nothing of importance, excepting there were many deaths from heart disease. At the age of twelve, he had pneumonia which was nearly fatal. At the age of fifteen, he suffered

from recurring attacks of rhinitis. At the age of twenty-five, he had socalled inflammatory rheumatism, affecting the left elbow which was red, swollen and painful. He has been under a series of nervous strains, has overworked more or less continuously, and is moderately obese.

The teeth show well marked periapical infection, the removal of which was followed by severe and persistent hemorrhage, and when examined, showed streptococci. The tonsils were diagnosed as infective, in April, 1922, and the crypts showed streptococcus hemolyticus and staphylococcus aureus hemolyticus, from which a vaccine was made and administered, for the reason that he declined operation.

Reexamination in December, 1922: The pulse in sitting position was 76, systolic pressure 155, diastolic pressure 100, pulse pressure 55, only slightly influenced by recumbency or exercise. Erythrocytes 4,490,000 or 90%, hemoglobin 85%, leucocytes 6,600, polynuclear cells 60.5%, large lymphocytes 8.5%, small lymphocytes 30%, mast cells 1%. The slight reduction in the leucocytes and the increase in the lymphocytes were believed to be due to tonsillar infection. The urine was concentrated, and contained a large amount of indican and oxalat of calcium crystals, a few cylindroids, but no albumin or sugar.

He complained of weakness, nervousness, numbness of both arms and hands, and a slightly enlarged finger joint. The joint of the middle finger of the left hand is not only enlarged, but painful.

Cardiac compensation for the old mitral valvulitis was fairly well maintained. The occurrence of arthritis, numbness, slight anemia, leucopenia and lymphocytosis, all indicated the entrance into the circulation of toxins or streptococci, or both, and the only focus of infection discoverable was the tonsils. The tonsils presented the appearance of two large, spongy masses, and each contained foul smelling secretions, and material from the crypts revealed an almost pure culture of hemolytic streptococci.

The operation was performed by Dr. Ross H. Skillern, January 9th, 1923, and the tonsils were so soft that they could be removed only piecemeal. As the hemorrhage was so persistent after the removal of the tooth, it was anticipated that hemorrhage would follow the removal of the tonsils, but this did not occur. The patient did well until January 13th,

four days after the operation, when a considerable nasal discharge occurred, accompanied by a rise of temperature to 103° F., pulse 114, respiration 26, and the urine now showed a trace of albumin and granular hyalin tube casts, and two days later, the albuminuria and cylindruria increased, due to an acute infective nephritis. The urine also contained a large amount of indican and skatol, indicating intestinal toxemia. On January 14th, Dr. Alexander Klein reported that a dry, hacking cough had developed, temperature 102.8° F., pulse 98, respiration 24. The nasal obstruction was well marked, and the upper lobe of the *right* lung showed signs of beginning consolidation. On January 15th, temperature 104° F., pulse 120, respiration 32, delirious, and the pneumonia had extended to the *base of the other lung*. On January 17th, temperature 102° F., pulse 140, respiration 40, semiconscious, unable to answer questions, pulmonary edema, rapidly failing circulation, and death occurred early the following morning, eight days after the operation.

REMARKS: The friable condition of the diseased tonsils necessitated removal piecemeal, favoring the absorption and aspiration of streptococci and toxins. The extension of pneumonia from one lung to the other and the acute infective nephritis, suggest virulency of the streptococcus or loss of resistance, or both. The previously damaged cardiovascular system permitted the circulation to yield more easily and favored pulmonary edema.

Although the patient was definitely substandard in health, we did not believe the risk from the tonsillectomy would be great, and the fatal pneumonia was not expected. It is regrettable that when the diagnosis of infected tonsils was made in May, 1922, he did not accept the advice that they be promptly removed.

A review of this case gives no data by which death may be anticipated or prevented under similar circumstances. If the virulency of the streptococcus, or the susceptibility of the patient had been determined before the operation, perhaps the prognosis would have been less favorable.

The remarks under Case II, are equally applicable to this case.

Many physicians and laryngologists fail to recognize the etiologic relationship of the removal of infected tonsils to pneumonia and pulmonary abscess. Pneumonia and pulmonary abscess follows tonsillectomy more frequently than is

generally believed, and may occur one or more weeks after the operation and, in such cases, the causal relation of tonsillectomy is frequently overlooked.

The following suggestions may be of advantage in cases similar to those reported:

(1) The preoperative local treatment of infected tonsils, although unsatisfactory, may give partial good results. The crypts are often closed by adhesions, and a retrotonsillar abscess cannot be favorably influenced by local treatment; nevertheless, when the mortality risk is high, the crypts should be emptied as far as possible by mechanical means, especially by suction. The division of the cryptal walls, so as to favor drainage, and secure more infected material by suction, and the local application of iodin is recommended.

(2) The operator should give serious thought to the especial requirements of the individual patient, so far as the anesthetic is concerned, and the operation should be adapted to the peculiar requirements of each case.

(3) In suitable cases, the Waring Suction Tube,[3] the Wieder[4] Cup, or the Moor's[5] Sump should be of value, when the mortality risk from tonsillectomy is large.

(4) Team work upon the part of the surgeon, the assistant, the interne, the anesthetizer, and the nurse.

(5) The surgeon should confer with the consulting physician before, during and after the operation.

(6) 10 c.c. of a 10% solution of calcium chlorid, in redistilled water, should be given intravenously for three days prior to the operation even though the blood coagulation time is normal, and is especially indicated in hemorrhagic cases.

(7) An autogenous vaccine before the operation will increase antibody resistance, lessen the operative risk, and expedite the recovery.

(8) Senile, asthenic and cardiac patients should receive the infusion of tinctur of digitalis, tinctur of nux vomica, and whiskey for one week before and after the operation.

(9) In the postoperative period, rest in bed, the judicious use of morphin, atropin, whiskey, digitalis, nux vomica and laxatives are indicated.

(10) After the operation, the patient should rest on the side, with the head turned so as to facilitate the escape of saliva and blood. Hawking, clearing the throat, and talking should be avoided.

BIBLIOGRAPHY.

1. Martin, G. Ewart. Jour. Laryng., Otol. and Rhinol., Feb., 1922, p. 80.
2. Fetterolf, George. A Case of Posttonsillectomy Pulmonary Abscess. The Laryngoscope, July, 1922.
3. Davis, J. Leslie. Tonsillectomy and Anesthesia. Therap. Gazette, March 15, 1923.
4. Wieder, Henry S. A Cup Device to Prevent Pneumonia Following Tonsillectomy by the Sluder Technic. Eye, Ear, Nose and Throat Monthly, Jan., 1924.
5. Moore, William Frederick. Tonsillectomy Sump. The Laryngoscope, Sept., 1922.

DISCUSSION.

DR. ROSS H. SKILLERN, Philadelphia, Pa.: I wonder if it has ever occurred to any of us to think of the attitude of the general laity towards an operation for removal of the tonsils. If a patient is sent to the hospital for removal of the appendix, the family is worried and everybody looks on it as a serious operation, and until it is over they are on edge. But a tonsillectomy in a child is looked upon as more or less of an everyday sort of matter—nothing possibly can happen—it is nothing that we need worry about; and although the laity look upon a tonsillectomy in an older person as something more serious, even under those circumstances, when the patient comes to the hospital, it is sort of a joking matter. But let something occur, let death occur after a tonsillectomy, it does not matter whether the patient cuts his throat, jumps out of a window or dies from pneumonia, to the family he is dead, and the operator is connected with the case. I will not say he is responsible, but he is more or less responsible, and his lack of skill is proclaimed from the house tops—something that many of us have lived through. I have, for one, and I can assure you it is a very unpleasant experience.

Now, we have many complications, such as hemorrhage, that we fear more than pneumonia, but pneumonia is the specter which is always lurking around the corner. How, then, can we prevent the patient from dying of pneumonia? It seems to me there are two times at which a patient may become infected with the pneumococcus. The first is during the operation, and the second is after the operation. During the operation, suppose the anesthetic is pushed to the third degree, where the reflexes of the pharynx and the larynx are lowered or perhaps lost. As Jackson has well put it, cough is the watchdog of the lungs, and every man here has undergone a certain feeling when a patient lying on the table under deep narcosis suddenly begins a suffocating, choking, gagging cough. Does he continue the operation? No, he stops to see what is causing the choking. At that particular time, there is the gravest danger of having the patient too deeply under narcosis in tonsillectomy. I think the pharyngeal reflexes should be lost, but not the laryngeal, which we all know are the last to leave.

It has often been said to me, "Don't you think the ether vapor apparatuses are dangerous, that they may force blood or infection into the larynx?" I think that is absurd. I do not know how we can blame any apparatus for anything of this sort, so I think we can wave that to one side. How will we prevent a patient aspirating blood and mucus? By using suction and keeping the posterior pharyngeal wall as clean as the palm of one's hand.

Another thing is that we usually operate in a pretty warm room—85° perhaps, or up to 92° and 93°. What happens frequently? A patient is not only exposed, but frequently he is taken into a cold corridor, inadequately covered up, and it is a wonder to me that we do not have a great deal more than a simple chilling of the body. In my case that Dr. Daland spoke of, I will tell you something that he does not know, which is that the evening following the operation, that man was notified by a pupil nurse in the hospital that he was wanted at the phone. He got out of bed in his night clothes, walked the length of a cold corridor, stood at the phone and conversed for five or ten minutes, and then went back and got into bed. In twenty-four hours there was a chill and a sudden jump in temperature. Whether that had anything to do with it, I do not know, but who would like to have a patient try that little experiment after a tonsillectomy? I think none of us. So we must be particularly careful about the exposure, even after seventy-two hours following such a thing as tonsillectomy. I sincerely hope, that those of you who have never been placed in a position of this sort will never be obliged to endure the painful experience of losing a patient after a tonsillectomy.

Dr. Harris P. Mosher, Boston: The speaker says that deaths from bronchopneumonia following tonsillectomy are rare. I do not know the number of cases we have had at the Massachusetts General Hospital, but I think about the usual number.

I would like to take up the conclusions of the speaker. He speaks of the preoperative cleansing of the tonsils. It seems to me there is something that none of us think about seriously, cleansing the tonsils as well as you can in any way you prefer. He also said that the operator should give serious thought to the special requirements of the individual patient, so far as the anesthetic is concerned, and the operation should be adapted to the peculiar requirements of each case. I think the first case should have been operated probably under local anesthesia.

Then he speaks of suction during the operation. I am in favor of suction if the apparatus is not so clumsy that it gets in the way. You can evacuate the tonsils thoroughly, I think, for practical purposes, by sustained pressure with a vacuum apparatus.

Now, he comes to something important, and that is team work upon the part of the surgeon, the assistant, the interne, the anesthetizer and the nurse. He explains that by saying that the operation should be as short as possible, and I agree with that.

Then he says the surgeon should confer with the consulting physician before, during and after the operation. I agree with that. Before the operation get all the medical advice you can, if they are not trying to push a surgical wreck upon you. I agree that we should have every aid that the medical man can give us after the operation, but what use is the physician during the operation? If you make him first assistant he simply prolongs the operation.

He says he uses 10 c.c. of a 10 per cent solution of calcium chlorid. It has been my practice to give calcium lactat as a routine a day or two before a tonsillectomy.

He uses vaccine. I have an open mind on that, but I have not had much success with it.

Then we come to his conclusion in regard to postoperative medical care, and I feel that is very important.

In closing, I would like to agree with Dr. Skillern, which is a pleasure; I would like to disagree, which is also a pleasure. His first question was whether the laity sufficiently appreciated the tonsil operation. It is a serious operation, even in children, and there is no operation in the adult that I dread more.

Now, about the ether machines mechanically controlled. I do not like them; I am afraid of them. Woman, glorious as she is, is not a mechanic. In many of our institutions a trained woman anesthetist is used. I am afraid to put a mechanical ether vapor machine into the hands of a woman. I will, however, endorse some simple form of mechanical vapor apparatus worked by the foot, but a woman anesthetist cannot be trusted to handle a machine which is controlled by a stop cock, because we have found in our part of the country, that she easily becomes mixed up as to which stop cock to turn.

Dr. Joseph C. Beck, Chicago: It certainly has been a pleasure to listen to the presentation of these three cases by Dr. Daland, and to the discussion of Dr. Skillern and Dr. Mosher—one serious and the other facetious. Now I must try to hit the middle line. In the first place, I would like to say that I have been very fortunate in having had no experience with either pneumonia or lung abscess following tonsil operation, either in clinical work or in my private practice, so from experience I should not speak at all. However, one of the striking points made by Dr. Mosher is true, and that is that the internist and the heart and lung specialist particularly, should work together with the oto-laryngologist. One I have in mind, in Chicago, who refers to me cases for removal of tonsils in adults for the cure of cardiac disease, and he insists on local anesthesia—in a patient who is already so low in vitality that you surely feel you should refuse to follow his advice, even though the man is prominent in that particular field. I have not refused to operate. I have in some cases delayed operation, playing for time, and in others where I did not want to operate under local anesthesia, I have given synergistic analgesia, which gives good results and obviates irritation from the anesthesia, and also obviates pneumonia or lung abscess.

I want to mention another point, and that is operation during epidemics, particularly influenza, when we do not know that the patient is having the incubation period of influenza. I have caught cases like that on the operating table. They have been given the anesthetic, and when I saw clear mucus coming from the nose, the operation ceased right there and was delayed. It is too bad that the patient receives any ether, but I feel I have done the best thing for him in not operating at that time.

Suction is a good aid, but you do not need the machine in your way. Have a suction machine in the basement and pipes leading to the table.

Dr. John F. Barnhill, Indianapolis, Indiana: In the first case reported by Dr. Daland, it seems reasonably certain that most of the trouble experienced came because of the profuse hemorrhage, and therefore any study of the case that could have been made previously by the physicians in charge, with a view of avoiding such difficulty, would have

been well worth while. Also, any plan of procedure that could have been chosen at the time of operation, that might have avoided the hemorrhage, would probably have eliminated the causes that led to the pneumonia and lung abscess. I do not infer from the paper that such precautions were not taken in this case, but only wish to emphasize the fact, that in certain decrepit persons, every preliminary investigation, precaution and preparation is necessary to safe tonsillectomy.

In cases that are not good operative risks, such as have been described, a good hospital organization is of great assistance to safe operation. A thoroughly trained anesthetist is of first value, because training and judgment on his part will enable him to administer a thoroughly satisfactory anesthesia with but a minimum of ether. A good organization enables the operator to remove the tonsils well, usually in but a few minutes, and this alone adds very much to the safety of the procedure.

In one of the cases reported, it seems to me a local anesthetic would have been preferable. However, I do not wish to appear a critic in any case which I have not personally examined.

I do not believe it can be truthfully said, that the proper use of suction apparatus has ever been responsible for pneumonia or lung abscess following tonsillectomy. These and other misfortunes sometimes result, as all must honestly admit, even when every precaution humanly possible has been taken to make the patient safe.

Very fortunately, in a rather long experience, I have had no case of pneumonia or lung abscess following tonsillectomy under a general anesthetic. I have not been so fortunate in one case following a local anesthetic. In this case, not one of all the reasons that have from time to time been assigned as a cause of lung abscess was clearly present. The patient, an adult, was reasonably well in every way, except occasional attacks of tonsillitis followed by persistent rheumatic attacks. The patient was under observation before operation sufficiently long to enable me to make thorough examinations and adequate preparation. Three drams of a 1 per cent solution of novocain, with 5 minims of adrenalin chlorid, were used to inject the peritonsillar space in the usual way. The removal of the tonsils was accomplished quickly, and without incident, there being neither bleeding nor pain. Three days following this apparently simple procedure, a sharp chill occurred followed by high temperature. A septic temperature with repeated chills followed, multiple abscesses of the lung could be made out, and death occurred in about ten days.

I can offer no helpful explanation as to the cause of this death. The operation was performed in a first class hospital, where every precaution as to sterility of instruments and of the fluid injected was assured. Such misfortune emphasizes the fact that he who operates much must expect difficulties occasionally, even though his team work be perfect and his technic reasonably good.

I fully agree with Doctor Daland that, everything considered, these patients should have been operated, for I have no doubt they represent but a few out of a large number of similar cases where recovery following tonsillectomy was prompt and wholly gratifying.

Dr. William G. Hepburn, Montreal: I would like to tell you about a method of intratracheal anesthesia which has been in use in the Montreal General Hospital for twelve years. During this period, we have not had a case of lung abscess or pneumonia following its use. Intratracheal anesthesia is used for our adult cases of tonsillectomy to the extent of about 99 per cent. We give intratracheal anesthesia to any patient that has good heart compensation. If there is poor heart compensation, we do not give any general anesthetic. Under intratracheal anesthesia, the pulse rate and respiratory rate are lower than with any other form of anesthesia. We give ether intratracheal anesthesia by means of a pressure machine. This machine delivers a stream of warmed ether under pressure into the trachea. I do not believe that any form of general anesthetic delivered by the oral or nasal method is a safe procedure.

By this method, the patient is placed under general anesthesia to the stage of relaxation, and then the intratracheal tube is introduced. The operator, therefore, has a quiet throat in which to operate; he can take his time in doing the operation, and keep the patient on the table for a sufficient length of time to check a hemorrhage. Fresh air is administered until the cough reflex has returned, and pharynx is rendered dry by suction before the tracheal tube is removed.

Dr. Arthur L. Weil, New Orleans: The case described by Dr. Daland is one type of case in which abscess may more or less be expected occasionally—a bad surgical risk—the patient advanced in years, and the patient with cardiac renal affection

There is another class of case in which we all fear an abscess of the lung, quite distinct from this class; healthy young adults and children in whom the operation is successful, quickly and uneventfully carried out, but in whom these complications develop. The question is how can we avoid these things? In the case of patients in advanced years, of course we can avoid it by not operating, but the others we have to operate. I had not seen such a case until recently, but it was my misfortune to take part in a tragedy—a young child about eight years of age, a beautiful, healthy, robust little girl, the sister of two brothers, all the children of a dentist. All the children had bad tonsils, but were otherwise healthy. These children were all operated the same day. The operation was easy in every case, no bleeding, no trouble with the anesthesia; the operation of the little girl lasted not over ten to twelve minutes, bad adenoids and tonsils being removed. These children had an uneventful convalescence, and on the eighth day the three were discharged in apparently perfect health, with the throat practically healed. Two days after that, this little girl developed temperature. I thought of intestinal disturbance, but the temperature persisted and in a week she became more or less septic. Having heard so much about pulmonary abscess, I began to suspect that might be the case. Examination at that time by the most skillful internist revealed nothing. We had an X-ray made, and in the upper lobe of the right lung there was a spot so small that the radiologists did not agree—one thought it was a beginning abscess and the other did not know. However, it went on until it was an unquestionable abscess, which was later opened and a large amount of pus drained. The child got along for a week or ten days, and then

began to show signs in the other lung. X-ray showed an abscess in the left lung, and when the abscess was opened, a large quantity of pus was evacuated.

Stress has been laid in the discussion upon the advantages and disadvantages of one form of anesthesia over another, and the possibility of the aspiration of blood and infected matter. When these cases of pulmonary abscess take place, as this one did, ten days or two weeks after operation, I do not believe the abscess is due to aspiration or to improper anesthesia, because if aspiration pneumonia or abscess follows, it should occur within two or three days after the operation is finished. There is something that has caused this condition other than aspiration, probably metastatic infection through the blood stream, or the lymph stream, probably the former, a secondary result of the trauma of the operation. It is something of that kind that causes these late appearing abscesses in cases otherwise healthy, and we must find out what it is if we are to keep these healthy patients from developing pulmonary abscess following tonsillectomy.

Dr. Judson Daland, Philadelphia (closing): In the first place, I want to thank the Fellows of the Academy for their discussion of this paper. I was much interested in the different points of view presented. I came here hoping to obtain some information, as I am not a laryngologist.

I do not know as to the persuasiveness of clinicians, but while listening to Dr. Mosher, I was convinced that he has more persuasive power than any internist I know. I desire to call Dr. Mosher's attention to the fact that "these persuasive internists, with their remarkable ability to use language, through which they get their living" are not really concerned so much about their own living as the living of the patient. Physicians frequently refer patients to me in the last stages of chronic disease. I do not pass them to the surgeon because I want to pass the buck. The one thought is to help the patient. I have seen a very large number of patients like those reported, suffering from constitutional infection, tonsillar in origin, regain a fair degree of good health and live many years after tonsillectomy, who otherwise would have died.

In Case I, Dr. Fetterolf and myself recognized the increased mortality risk, but did not consider the patient inoperable. In Case III, Dr. Skillern and myself were of the opinion that the patient was operable, despite the increased mortality risk. The anticipated hemorrhage did not occur, and the old mitral lesion probably played a minor role in the occurrence of pneumonia. In other words, in complicated cases of cardiovascular diseases, in association with a tonsillar infection, if the infection is not removed the patient dies, and if removed, recovery usually follows.

A tonsillectomy should never be performed excepting with the full consent of the patient and family, after the dangers of the operation, and of allowing tonsillar infection to continue, have been fully explained. It seems to me that Dr. Mosher disposes of these complicated cases altogether too easily by stating that they are inoperable.

What I most desire from the Fellows of the Academy is definite information regarding surgical procedure and the anesthetic in advanced cardiovascular disease complicated by tonsillar infection. The decision as to which form of anesthesia is best for these complicated cases should be in the hands of the laryngologist. I find, that in one city general anesthesia is almost the universal rule, while in another local anesthesia is the general rule. It seems to me that the laryngologists should come to some agreement as to what is best in these cases.

The only reason that mention of suction in tonsillectomy was made in my paper was for the purpose of discussion. It seems to me, that a mass of diseased tonsillar tissue containing virulent streptococcus hemolyticus should be quickly withdrawn from the throat, so as to lessen the possibility of postoperative pneumonia or abscess.

THE CILIARY INHIBITION OR DESTRUCTION IN TRACHEOBRONCHIAL ASTHMA, WITH NOTES ON BRONCHOSCOPIC TREATMENT.

WILLIAM F. MOORE, M. D.*

PHILADELPHIA, PA.

In again bringing this much discussed disease to your attention, our object is to present results of bronchoscopic, histologic and bacteriologic studies in a series of cases from the Bronchoscopic Clinic of the Jefferson Medical College Hospital of Philadelphia.

A majority of these cases are chronic forms of many years duration and marked severity. They have passed through the Asthma Clinic, where full sensitization tests have been completed and treatment given. Many have been studied thoroughly in the other special clinics at the Hospital.

Theories as to the causative factors which are thought to produce attacks of tracheobronchial asthma are legion, and time and space do not permit of their enumeration. One is impressed on reviewing the clinical material, and on bronchoscopic examination, with the fact that we are not dealing in each case with the same conditions.

The speedy relief in the great majority of our cases has led us to believe that bronchoscopy alone, affording as it does a means of cleansing the overloaded mucous membrane of the tracheobronchial tree, temporarily alleviates the symptoms and yields relatively permanent results in many cases. This would seem to justify the conclusion that defective ciliary drainage is a factor of the utmost importance.

The question of primary or secondary infection of the bronchi is of great importance. This is difficult of solution. Our help in this must come from the internist and specialist, who can point out the possible source.

Primary cases have responded permanently, but secondary ones require, of course, eradication of or treatment at the source.

---

*Bronchoscopist, Department of Diseases of Chest, Jefferson Hospital.

TECHNIC.

Asthmatic patients referred for diagnostic bronchoscopy and treatment are first given a complete physical examination by the internist. Roentgen ray studies are then made. As the majority of these cases have come through the Asthma Clinic, sensitization tests have been completed. No patient is bronchoscoped without having had a physical examination and roentgen ray studies.

At the first examination, morphin is generally administered hypodermatically in doses of from ⅛ to ¼ grain, one hour before bronchoscopy. No general anesthetic has ever been found necessary. In children, no cocain is used, but in adults a 10 per cent solution is applied deep enough in the pharynx to inhibit the superior laryngeal nerve (Moore[2]). This is applied by means of a swab on a curved applicator forceps, and repeated once.

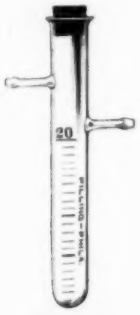


Fig. 1.

There is no contraindication to a bronchoscopic examination during an asthmatic attack, in the type of cases herein referred to. Preliminary medical examination has usually eliminated socalled "cardiac asthma" and other conditions in which there is no indication for bronchoscopy.

BACTERIOLOGIC SPECIMENS: Specimens are obtained bronchoscopically, uncontaminated by oral secretion, by two means; either by swab or, where the secretion is not too viscid and is susceptible of aspiration, it is drawn into specially devised tubes (Fig. 1) which are inserted between the aspirating bronchoscope and the rubber tube from the suction pump. The tubes are sealed for laboratory examination later. Specimens

are immediately "planted" on human blood agar-agar, and slide smears made by George R. Moffitt, one of the group, who will present the results of his work in the discussion which is to follow and, more fully, in an independent paper[3]. An abstract of some phases of his work is given in a subsequent paragraph.

Bronchoscopic Treatment: Two methods are employed to rid the mucous membrane of the trachea or bronchi of their overload of secretion; namely, sponging where the secretion is thick and tenacious, or aspiration, either through the special aspirating bronchoscope or with a separate aspirating tube inserted through and beyond the bronchoscope. Forceps are ordinarily not required for this. A solution containing 10 drops of a 10 per cent solution of cocain, 20 drops adrenalin solution (1-1000), in one fluid ounce of normal saline as a vehicle is then instilled, using 5 c.c. in each main stem bronchus. A 10 per cent solution of silver nitrat has been applied to localized areas in certain cases, and in others, guiacol in 5 per cent and gomenol in 20 per cent strength have been used. However, the adrenalin-cocain combination above mentioned, which we know as the "Asthma Solution," is the one most frequently employed.

Hospitalization is not required, and the number and frequency of treatments generally depends, after the first two bronchoscopies, on the character and frequency of the asthmatic attacks, the patient being instructed to report at weekly intervals, so that accurate data may be kept. Some patients require but three or four bronchoscopic treatments, while others have needed as many as eighteen.

Vaccines.

One week after the diagnostic bronchoscopy (at which specimens are taken), the autogenous vaccine is ready for administration. This is given at four day intervals in ascending dosage, as outlined by Dr. Moffitt. This part of the treatment is either carried out by the physician referring the case, or the patient returns to the Asthma Clinic for administration.

Bacteriology and Vaccines: Among the various organisms, the streptococcus viridens, streptococcus hemolyticus, or a hemolytic strain of the staphylococcus aureus, in pure culture or associated, are conspicuously present in nearly all cases of asthma. Their role may be (a) causative factors, or (b) secondary invaders responsible for a primary bronchitis upon

which the asthma develops. It is reasonable to believe, and this belief is supported by our results, that the use of autogenous vaccines prepared from the organisms found would give more promise of establishing, as permanent, the relative cures which have been effected by local treatment, especially by the assistance of ciliary drainage to be accomplished by bronchoscopic aspiration.

The organisms recovered from the secretions of asthmatics, removed by the bronchoscope without oral contamination, show a marked degree of attenuation, which may acount for the nonoccurrence of metastatic infection. It would, however, not render less potent vaccines prepared from them; for loss of virulence of an organism does not, in any way, effect the efficiency of the vaccine.

Classification of Cases.

From the standpoint of results obtained, it has seemed logical to assume that the twenty-seven cases herein reported should be grouped in a broad, but convenient, classification, as: (1) Those with bronchoscopically evident active suppurative tracheobronchitis[1]; (2) Those with a bronchoscopically evident chronic passive congestion. In the first group are placed all those showing an inflammatory mucosa with a great deal of secretion. In the latter are placed those cases in which the mucosa has assumed the bluish-red, leathery appearance, with little or no secretion, or those of inspiratory or expiratory collapse, also with little secretion. It has seemed to us that a true infection was responsible for the major (first) division, while in the second group, factors outside the tracheobronchial tree were responsible for a continuation of the symptoms; and the local infection if any, was secondary and played a minor role in causing attacks. We are cognizant of the fact that this division is open to criticism from many angles, and that the border line is rather hazy in some cases; still, it is necessary for workers in this field to have some criteria for classification that may serve as a basis for therapeutic indications from which they may reasonably expect to get results.

Class 1—Tracheobronchitis Present.

Case Number	Result
3	Great improvement. No attacks in 8 months.
4	Improved.

5	Improved.
6	Improved.
9	Marked improvement.
10	Improved. No attacks in 4 months.
11	Improved.
12	Greatly improved.
13	Greatly improved.
14	Improved.
15	Improved
16	Improved. No attacks in 2 years.
17	No change.
19	Pansinusitis. Referred for diagnostic bronchoscopy only.
20	For diagnostic bronchoscopy only.
21	Attacks decreased in severity and frequency.
22	Improved. One attack in four weeks.
23	Improved. No symptoms for 1 month before discharge.
26	Improved.
27	For diagnostic bronchoscopy only.

CLASS 2.—TRACHEOBRONCHITIS ABSENT.

Case Number	Result
1	Temporary relief.
2	Temporary relief.
7	No relief. Probably due to a postpneumonic suppuration.
8	Unimproved. Diagnosis: Cardiorenal disease.
18	No change.
24	Temporary relief only.
25	Referred for treatment for pansinusitis.

REPORTS OF CASES.

In presenting the following tabulation of cases, it will be noted that none has been pronounced cured, although two years have elapsed in one case since having an attack. When a case can be pronounced permanently cured seems justly to be a much mooted question with the internists. It would seem that for the purposes of determination of the efficiency of treatment, a patient free from attacks for a year might be called a relative cure.

CASE 1. Duration of disease: 20 years.

Appearances at diagnostic bronchoscopy: Bronchial mucosa inflamed and velvety. Small amount of secretion present.

Report of roentgenologist: Peribronchical thickening; increased density of root shadows.

Solution employed: Cocain-adrenalin.

Second bronchoscopy: Tracheobronchial tree found normal.

Bacteriology: Streptococcus viridens.

Number of bronchoscopies: two.

Result: Improved. Has not returned for treatment.

CASE 2. Duration of disease, 3 years.

Appearances at diagnostic bronchoscopy: Inflammation localized; collapse of posterior wall of trachea; very little secretion.

Report of roentgenologist: Thickening at roots of both lungs. Heavy peribronchial thickening in right lower lobe.

Endobronchial application employed: Cocain-adrenalin solution, and silver nitrat.

Number of bronchoscopies: four.

Result: Improved. Has not returned for treatment to date.

CASE 3. Duration of disease, one year.

Appearances at diagnostic bronchoscopy: Trachea intensely injected. Left bronchus normal in color, form and size of openings. No granulations. No patches of abnormal mucosa. Right bronchus inflammatory. Mucous secretion visible throughout all bronchi of right lung. On inspiration, mucosa pulled out into strings across mouth of bronchoscope. All orifices inflamed and have pus clinging to walls; diameter normal. Right bronchus dilated.

Report of roentgenologist: Marked thickening of roots of lungs, both sides. Peribronchial thickening left lower lobes and gland shadows are seen at lower border hilus. Shadow right side.

Endobronchial medication employed: Gomenol; cocain-adrenalin.

Number of bronchoscopies: 15.

Result: Discharged with no symptoms, and has continued so for 10 months.

Bacteriology: Streptococcus viridens; streptococcus hemolyticus; bac. influenzae.

CASE 4. Duration of disease: Nine years (daily attacks).

Appearances at diagnostic bronchoscopy: Bronchial mucosa chronically inflamed; walls do not have bluish-red

appearance seen in cases of passive congestion; tenacious, scant secretion clinging to walls, and stretching across lumen as strings.

Report of roentgenologist: More than average thickening in right lower lobe along the main bronchi.

Endobronchial application used: Cocain-adrenalin.

Number of bronchoscopies: 4.

Result: Improved when last seen in Dispensary 15 months ago. Seen 12 months ago by social worker, who reported patient had not suffered from asthma lately.

Case 5. Duration of disease: 9 years (severe).

Appearances at diagnostic bronchoscopy: Mucous membrane has the characteristic bluish-red color; passive congestion. Not enough exudate to aspirate into receiving flask. Specimens taken by swab for culture.

Report of roentgenologist: The right upper and middle lobes are fairly dense and markedly granular in appearance, and give the impression that they are literally studded with tubercles throughout.

Solution employed: Cocain-adrenalin.

Number of bronchoscopies: 5.

Result: Improved.

Case 6. Duration of disease: 5 years.

Appearances at diagnostic bronchoscopy: Trachea shows some collapse on expiration. Mucosa of both main stem bronchi inflamed and covered in patches with a thick and, in some areas, a dry crust like mucopurulent material.

Report of roentgenologist: Root shadows are heavy and lungs are emphysematous.

Solution used: Cocaine-adrenalin after suction cleansing.

Number of bronchoscopies: Seven.

Result: Marked improvement. Died of intercurrent disease. Two attacks since beginning treatment.

Case 7. Duration of disease: 15 years (every winter).

Appearances at diagnostic bronchoscopy: Trachea and bronchial mucous membrane congested and appear somewhat edematous. Just above the carina, there is a thick patch of white secretion tightly adherent to wall on right side. Again, below this, is another similar patch. Purulent material exudes from all bronchial orifices right side. On removing patches, eroded mucous membrane is exposed.

Report of roentgenologist: Considerable fibrosis in lower half of right lung, and abnormal elevation of right diaphragm. This has the appearance of a postpneumonic process.

Solution used: 4 c.c. 20 per cent argyrol.

Number of bronchoscopies: two.

Result: Unimproved. Unsatisfactory case on account of poverty and resultant home conditions.

CASE 8. Duration of disease: one year.

Appearances at diagnostic bronchoscopy: Mucous membrane of trachea cyanotic. Bluish-red, velvety, appearance. Mucous membrane appears thickened. No secretion, except a very small amount from left lower lobe bronchus on forceful coughing.

Report of roentgenologist: Considerable fibrosis in both bases extending from the hilus zones. Marked peribronchial thickening throughout both upper lobes. Bronchiectatic cavities in hilus region.

Number of bronchoscopies: four.

Result: Improved. Referred to Medical Ward, Jefferson Hospital, for treatment of myocarditis, and has not been seen since.

CASE 9. Duration of disease: 2½ years.

Appearances at diagnostic bronchoscopy: Mucosa of entire tracheobronchial tree inflamed. Tenacious grayish-white patches of mucus, which was obtained for bacterial study. The appearance was that of a chronic plastic tracheobronchitis.

Report of roentgenologist: There is considerable thickening at the roots of the lungs. No other evidence of pathology.

Bacteriology: Streptococcus viridens and staphylococcus aureus (hemolytic).

Number of bronchoscopies: eighteen.

Solution used: 2 per cent gomenol, and, finally, cocain-adrenalin.

Result: Improved; no attacks in 4 months.

CASE 10. Duration of disease: 15 years (very severe).

Appearances at diagnostic bronchoscopy: Mucous membrane of entire tracheobronchial tree injected and of a bluish-red tinge. Secretions quite viscid.

Report of roentgenologist: Except for a few fairly dense lymphatic glands in the region of the posterior wall of the main stem bronchus near bifurcation, no pathology.

Bacteriology: Hemolytic streptococcus.

Solution used: Cocain-adrenalin; 5 per cent guiacol once.

Number of bronchoscopies: twelve.

Result: Improved.

CASE 11. Duration of disease: three years.

Report of diagnostic bronchoscopy: Trachea and bronchi inflamed. Secretion present throughout trachea and bronchi.

Report of roentgenologist: Slight thickening at the roots of the lungs; otherwise normal.

Solution used: cocain-adrenalin.

Number of bronchoscopies.

Result: Improvement.

CASE 12. Duration of disease: two years.

Appearances at diagnostic bronchoscopy: Mucous membrane does not present the bluish color encountered in some of the other cases. Secretion present, encountered about ⅔ distance to carina (very thick). All bronchial orifices normal.

Report of roentgenologist: Marked thickening at roots of lungs, particularly on right side. Lungs greatly congested on both sides.

Bacteriology: Streptococcus viridens; streptococcus hemolyticus.

Solution used: 10 per cent silver nitrat applied locally; cocain-adrenalin.

Number of bronchoscopies: five.

Result: Greatly improved.

CASE 13. Duration of disease, two years.

Appearances at diagnostic bronchoscopy: Mucous membrane of trachea apparently normal. Thick viscid secretion coming from middle lobe bronchus right side. The upper lobe bronchus free and apparently normal. Lower lobe bronchi free and normal. Left side normal. Orifices normal.

Report of roentgenologist: Peribronchial thickening and evidence of bronchiectasis right lower lobe. On second examination, this had cleared considerably, but there was more evidence of pathology in the upper lobes (peribronchial thickening and scattered fine deposits in the parenchymal portion).

Solution used: Cocain-adrenalin. In this case K. I. was also administered.

Number of bronchoscopies: sixteen.

Result: Improved. No tubercle bacilli were found.

CASE 14. Duration of disease: 2 years.

Appearances at diagnostic bronchoscopy: The mucous membrane of trachea covered, especially on the posterior wall, with thick tenacious secretion. Caliber of right main stem bronchus diminished in size. No other abnormalites noted,

except that mucous membrane throughout tracheobronchial tree is injected.

Report of roentgenologist: Peribronchial thickening throughout.

Bacteriology: Sterptococcus viridens.

Solution used: Cocain-adrenalin.

Number of bronchoscopies: three.

Result: Improved.

CASE 15. Duration of disease: three years.

Appearances at diagnostic bronchoscopy: Mucosa edematous entire length of tracheobronchial tree. Active hyperemia, the mucous membrane having a red, injected appearance, rather than purplish. Large amount of sticky, mucopus adherent to walls and stringing from side to side in front of bronchoscope.

Report of roentgenologist: There is quite marked thickening at the roots of both lungs, and marked peribronchial thickening radiating in all directions from the root area on both sides.

Solution used: Cocain-adrenalin.

Number of bronchoscopies: two.

Result: Improved.

CASE 16. Duration of disease: twelve years.

Appearances at diagnostic bronchoscopy: On expiration, posterior wall of trachea collapses and approximates anterior wall. Bronchi narrowed probably due to spasm. Mucosa red and edematous and covered with thick white tenacious mucus.

Report of roentgenologist: Considerable thickening at roots of both lungs and dense calcareous glands on right side. Peribronchial thickening rather evenly distributed throughout chest, and of no definite characteristic appearance.

Solution used: Cocain-adrenalin.

Number of bronchoscopies: two.

Result: No attack in two years.

CASE 17. Duration of disease: 17 years (severe).

Appearances at diagnostic bronchoscopy: Tracheal mucous membrane covered with thick, tenacious mucopus. Pus present in right main stem bronchus. Mucosa of trachea and right main stem bronchus inflamed. (Much more marked in latter.)

Report of roentgenologist: Considerable thickening at roots of both lungs, and a fairly general peribronchial thickening, more marked in mid portion of chest.

Solution used: Cocain-adrenalin.

Number of bronchoscopies: two.

Result: No change.

CASE 18. Duration of disease: 10 years (very severe).

Appearances at diagnostic bronchoscopy: Trachea clear down to bifurcation. Mucosa red and inflamed and somewhat dry; at the bifurcation grayish, gelatinoid material encountered. Removed with difficulty.

Report of roentgenologist: None in history.

Number of bronchoscopies: one (for diagnosis).

Result: No change.

CASE 19. Duration of disease.

Appearances at diagnostic bronchoscopy: Mucosa of trachea and bronchi inflamed and velvety. Somewhat edematous. Layer of plastic secretion covering entire tracheobronchial tree.

Report of roentgenologist: Considerable thickening at roots of lungs and extension of peribronchial thickening, and probably bronchiectasis in lower lobes.

Solution used: Cocain-adrenalin.

Result: For diagnosis only; Pansinusitis.

CASE 20. Duration of disease: All life (39).

Appearances at diagnostic bronchoscopy: Very small amount of thin, elastic secretion over trachea and bronchi; pushes ahead in rolls in front of bronchoscope.

Report of roentgenologist: Peribronchial thickening along vertebral border of both upper lobes.

Bacteriology: Streptococcus viridens.

Solution used: Cocain-adrenalin.

Number of bronchoscopies: one.

Result: For diagnosis only; vaccine shipped. No further report available.

CASE 21. Duration of disease: 18 years (1 to 5 a day).

Appearances at diagnostic bronchoscopy: Mucosa of tracheobronchial tree shows patchy areas of inflammation, particularly marked at the carina, at the level between the left upper lobe orifice and the beginning of the left stem bronchus. On the right side, these patches entered down to the small bronchi. Very slight amount of thin, tenacious mucus, especially in these inflamed areas. Carina somewhat broadened.

Report of roentgenologist: Peribronchial thickening vertebral border right upper and lower lobe. Slight degree bronchiectasis left lower lobe.

Bacteriology: Staphylococcus aureus (hem.).

Solution used: Cocain-adrenalin.

Number of bronchoscopies: nine.

Result: Improvement. Attacks decreased from one daily to two and three a week.

CASE 22. Duration of disease: ten years.

Appearances at diagnostic bronchoscopy: Secretion very thick and tenacious throughout trachea, rolling up in such quantities about the mouth of the bronchoscope as to obscure view; too thick for aspiration.

Report of roentgenologist: Considerable peribronchial fibrosis both upper lobes, and I believe some degree of bronchiectasis.

Bacteriology: Streptococcus viridens and streptococcus hemolyticus.

Solution used: Cocain-adrenalin.

Number of bronchoscopies: seven.

Result: Improvement. The only attack since beginning treatment was during week treatment was omitted.

CASE 23. Duration of disease: four years.

Appearances at diagnostic bronchoscopy: Mucosa cyanotic and edematous. Pus seemed to be exuding from all bronchi. Tracheobronchial collapse on expiration.

Report of roentgenologist: Marked peribronchial thickening and fibrosis throughout entire chest.

Bacteriology: Streptococcus viridens; streptococcus hemolyticus; micrococcus cinerens.

Solution used: Cocain-adrenalin, 2 per cent guiacol, and 20 per cent gomenol.

Number of bronchoscopies: seventeen.

Result: Discharged to work. No symptoms since May 7, 1924.

CASE 24. Duration of disease: five years.

Appearances at diagnostic bronchoscopy: Bluish-red color of mucous membrane was characteristic. Present, however, only in carinal region. Mucus present only high in trachea.

Report of roentgenologist: Rather marked thickening at the roots of both lungs.

Solution used: Cocain-adrenalin.

Number of bronchoscopies: two.

Result: Improved.

Case 25. Duration of disease: many years.

Appearances at diagnostic bronchoscopy: Thin, mucoid secretion covering mucous membrane of trachea and bronchi. Mucosa inflamed and of a bluish tinge, and edematous. Bronchi considerably narrowed due to edema.

Report of roentgenologist: not available.

Solution used: Cocain-adrenalin and silver nitrat.

Number of bronchoscopies: six.

Result: Unsatisfactory. Referred for treatment of pansinusitis, but improved under treatment.

Case 26. Duration of disease: many years.

Appearances at diagnostic bronchoscopy: Mucous membrane inflamed, but does not have discorded, bluish, passive congestion of some of the cases. Mucous membrane is covered with thick, tenacious mucus.

Report of roentgenologist: Considerably more than average thickening at roots of lungs.

Solution used: Cocain-adrenalin; 10 per cent silver nitrat locally.

Number of bronchoscopies: thirteen.

Bacteriology: Staphylococcus aureus (hemolytic); micrococcus cinerens.

Result: Improved.

Case 27. Duration of disease: 2 years.

Appearances at diagnostic bronchoscopy: Mucosa of trachea and bronchi inflamed and edematous. No marked collapse on expiration.

Report of roentgenologist: Root structures rather heavy on both sides. Fibrosis left lower lobe, right middle and lower lobe.

Solution used: none.

Number of bronchoscopies; for diagnosis only.

Result: Not treated.

Ciliary Movements and Histology of Ciliated Cells.

Dr. George R. Moffitt has made a most interesting and important report of the results of his work with us at the Bronchoscopic Clinic in a paper on the "Pathologic Histology and Bacteriology of Asthma." With his permission we here present some of his observations which are pertinent to our subject:

The bronchial columnar epithelial cells are 40 to 50 micra

long, and 10 to 14 micra in diameter. Together with these cells are ciliated spherical cells, which do not appear to be the goblet cells that are normally present in the bronchial mucosa.

There are as many as 480 cilia attached to a cell. Works on histology have given the number as 12 to 25. The ciliary movements are at the rate of 360 a minute or six a second. There is a secondary ciliary movement, which forces secretion and foreign particles from the bronchial wall into the lumen. The cilia are 18 to 20 micra long. The only reference found upon this subject gives three to five micra as their length.

The basal or root process (not mentioned by any treatise on histology) apparently connects the cells of the free edge or uppermost layer to the basement membrane.

One or more of the following changes occur in the ciliated columnar epithelial cells of the asthmatic: (1) Loss of the ciliary motion which produces a current. (2) Complete loss of ciliary motion. (3) Actual loss of the cilia. (4) Fatty degeneration.

Regeneration of Ciliated Epithelium: The question arises as to the character of regeneration of the destroyed epithelial cells and repair of the injured bronchial mucous membrane. It is a well known pathologic fact that mucous membrane, in general, regenerate more rapidly and more perfectly than any other tissue. At the same time, it is uncertain as to whether a cell from which the cilia have been removed by trauma or disease could replace those lost processes by new ones.

There seems to be no reason why this should be considered impossible, and our observations lead us to believe that replacement of cilia or ciliated cells, lost by disease, with new cilia or new ciliated cells does take place. The cilia, as has been stated, are connected with delicate intracellular fibrillae within the superficial parts of the cell, and are in all probability continuous with these fibrillae, each of which, in turn, is connected with and controlled by two minute bodies of centrosomic derivation. It is not inconceivable that, when a cilium is lost, these bodies may cause the fibrillum to grow and extend beyond the cuticula as a new cilium. This would be a repetition of the process that goes on in each normal nonciliated cell as it reaches the free surface of the stratified layer of cells, for the cells beneath the surface as has been previously stated, possess no cilia.

However, if it is true that lost cilia cannot be replaced by

new ones, the general process of repair of the bronchial mucosa would not be delayed, for the injured surface cells would soon be exfoliated, as are all surface cells of stratified epithelium, squamous or columnar, and their places taken by the underlying cells, which would acquire their cilia by the ordinary physiologic process, whatever that may be.

If the destructive process were deeper than the epithelium, involving the basement membrane and tunica propria, an ulcer would result, and the process of repair would involve the formation of granulation tissue, new fibrous tissue and the proliferation of new epithelial cells from the uninjured cells at the edge of such ulcer.

Deep ulceration, however, as has been repeatedly demonstrated by bronchoscopy and autopsy, does not occur in asthma.

Conclusions.

1. Practically all cases which have come for treatment have shown, on roentgengraphic study, peribronchial thickening to a marked degree in some part of the lung; in some cases, general.

2. Those cases associated with a demonstrable tracheobronchitis have responded best to bronchoscopic treatment.

3. Ridding the tracheobronchial mucous membrane of secretion alone will temporarily benefit all cases.

4. While temporary relief follows these bronchoscopic treatments, permanent relief necessitates intrabronchial medication, the administration of vaccines made from the organism recovered directly from the bronchial secretions, and a general attention to hygiene and an abundant diet, the same as for tuberculous patients.

5. Regeneration of the ciliated epithelium occurs very rapidly after the secretion which has inhibited its action has been removed either by aspiration or swabbing.

6. Impairment of ciliary action has been conclusively demonstrated. Its restoration is imperative for lasting improvement.

BIBLIOGRAPHY.

1. Jackson, Chevalier. Suppurative Diseases of the Lung; Bronchoscopic Drainage as an Aid to Treatment by the Internist. Transactions of the American Academy of Ophthalmology and Oto-Laryngology, 1923.
2. Jackson, Tucker, Clerf, Lukens and Moore. Bronchoscopy as an Aid to the Thoracic Surgeon. Journal Amer. Med. Assn. In print. To be published.
3. Moore, William F. Pulmonary Abscess. Journ. Amer. Med. Assn., April 29, 1922, vol. LXXVIII, p. 1279.
4. Moffitt, George R. Bacteriology and Studies of Ciliary Motion. Tracheobronchial Asthma. In print.

OBSERVATION ON FIFTY CASES OF FOREIGN BODY IN THE AIR AND FOOD PASSAGES.*

GABRIEL TUCKER, M.D.†

PHILADELPHIA, PA.

The time has passed when the lodgment of a foreign body in the air and food passages is considered as a curiosity of medicine. (McCrae.) The monumental work of Chevalier Jackson[1] in his observations on the pathology produced by the lodgment of foreign bodies, and his untiring and self sacrificing efforts not only to develop the greatest skill and the highest perfection of technic in their endoscopic removal, but also to accumulate symptomologic data and develop diagnostic means, have awakened the medical world to the possibility of foreign body as an etiologic factor in disease, and its presence a possibility for exclusion in all cases of acute and chronic disease of the lower air passages and of the esophagus. The technic that make possible bronchoscopy and esophagoscopy without anesthesia, or in cases of bronchoscopy in adults, with local anesthesia only, enables both the internist and the surgeon to avail themselves of diagnostic and therapeutic means that are invaluable, and permits the removal of foreign bodies through the natural passages without resort to external operation.

In the recognition of the presence of foreign bodies in the air passages by physical signs, Professor Thomas McCrae, in his Lumleian Lectures[2], has given to the profession diagnostic signs which will enable the practitioner to make an accurate diagnosis in a very large percentage of the cases. In the roentgen ray diagnosis, Dr. Willis F. Manges[3], by original observations on atelectasis, and by using the phenomena of obstructive and compensatory emphysema, as first described by Iglauer, as the basis of study, has developed methods of

---

*From The Bronchoscopic Clinics, Philadelphia.

†Associate Professor of Bronchoscopy and Esophagoscopy, Graduate School of Medicine, University of Pennsylvania, Instructor in Bronchoscopy, Jefferson Medical College.

roentgen ray study in nonradiopaque foreign bodies, that definitely localize them in almost 100 per cent of the cases.

Many cases come to the physician with no history of foreign body accident, in which the presence of foreign body should be considered. Of these cases, may be mentioned recurrent winter bronchitis in children, pulmonary abscess, tuberculosis, thymic asthma, bronchiectasis, adenopathy, laryngeal stridor, dysphagia, regurgitation, and cylic vomiting. In cases supposed to have these conditions, we have found a foreign body as the cause of what seemed typical symptoms of these diseases, and its removal at the Bronchoscopic Clinic has cured the patient.

General physical examinations, mirror laryngoscopy, and roentgen ray studies should be routine procedures in all suspected foreign body cases. The characteristic physical signs of foreign body in the air passages are: the "asthmatoid wheeze", "audible slap", and palpatory thud (tracheal) of Jackson; the "tracheal flutter" and "limitation of expansion on the affected side" of McCrae; and the physical signs of obstructive emphysema, atelectasis, and drowned lung which arise from the mechanical obstruction to the bronchus by the foreign body and the inflammatory reaction which it produces. The careful consideration of these signs has enabled us on many occasions to make a positive diagnosis of the presence of foreign body, which was later confirmed by roentgen ray studies and bronchoscopic removal of the foreign body.

The roentgen ray study should include the air and food passages from the nasopharynx to the tuberosities of the ischia. It is only by taking this precaution, that the presence of foreign body can be excluded and a negative opinion given. A foreign body that was in the pharynx or esophagus may have been regurgitated into the nasopharynx, or it may have passed downward into the gastrointestinal tract.

Nonradiopaque foreign bodies may be outlined in some instances by the administration of an opaque mixture, preferably an aqueous solution of bismuth, or in case of fish bone, a capsule containing bismuth which will lodge at the level of the foreign body until it is dissolved. In many instances, the capsule will also stick after the foreign body has been removed, owing to the local esophagitis and spasm excited by the passage of the capsule, but it will pass on with the first or second subsequent swallow of water. If the foreign body

is high in the cervical esophagus, the capsule may pass by it with only slight hesitation, as was noted in a case (No. 13) reported in this paper.

The practitioner should guard against being misled in his interpretation of the signs and symptoms of foreign body, as a foreign body in the esophagus may produce evidences of respiratory trouble. An esophageal foreign body may produce dyspnea due to tracheal pressure; it may cause retention of secretion in the pharynx, and, with overflow and aspiration of the secretions, tracheobronchitis and aspiration bronchopneumonia may develop, though true lobar pneumonia is exceedingly rare even in case of foreign body aspiration. The foreign body may, from the inflammatory reaction, produce impairment of the motility of the larynx, and laryngeal stridor may result. In a case in the Clinic, a child, one year of age, on infantile liquid diet chiefly, had no particular difficulty in swallowing, but developed a stridor, which became worse, with increasing dyspnea over a period of three months. An otolaryngologist was consulted, and advised the removal of the adenoids. This was done under ether anesthesia, the child becoming very dyspneic while under the anesthetic. Stridor was not relieved by the removal of the adenoids, and Dr. H. C. Carpenter advised roentgen ray examination to exclude foreign body. This was done by Dr. H. K. Pancoast, and a flat, metallic foreign body was found in the cervical esophagus. The removal of the foreign body cured the stridor. Again, an esophageally lodged foreign body may ulcerate through the party wall, and the patient develop bronchial symptoms from the leakage.

The first point to be made, is that foreign body should always be thought of in case of a sick child; and the second point, is that only by the most careful study of signs and symptoms and the most expert roentgenologic study, with endoscopic examination of both air and food passages, is a foreign body to be excluded, and a differential diagnosis made.

The problems of endoscopic removal are many, and depend upon the age and size of the patient, the nature and conformation of the foreign body, its position, its location, etc.; and their solution is of the greatest importance in the safe removal of the foreign body. The information about the foreign body itself is best obtained by securing, when possible, a duplicate of the foreign body. The exact localization by physical signs

and X-ray study, and the duplication of the foreign body by the roentgenologist is also valuable. In order to meet the problems of endoscopic removal, proper equipment is the first essential. The second essential factor is a working team that has been drilled in the proper technic; one man without trained assistants cannot do good bronchoscopic work. It is essential that the assistant should be thoroughly trained in all phases of the work, and especially he should have a thorough understanding of the fundamental value of the position of the patient, and its most important relation to the problems of endoscopic removal of foreign bodies. The Clinics doing the

CASE REPORTS.

best endoscopic work will always be those where two or three endoscopists work harmoniously together.

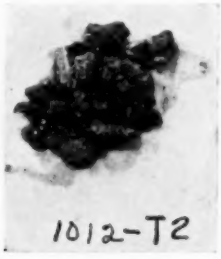
The cases here reported are from the Bronchoscopic clinics of Chevalier Jackson, at the University of Pennsylvania Hospital and Jefferson Hospital, Philadelphia.

All of the foregoing assertions are based upon observations in the cases recorded below; additional points will be commented upon apropos of the individual cases.

Many of the cases here reported were tried unsuccessfully by various bronchoscopists elsewhere before admission, but for obvious reasons this has been left out of the record.

CASE 1. Foreign body. 943T1. *Bolus of Meat in the Esophagus. Cicatricial Stenosis of the Esophagus.* Colored woman, aged 26, had choked while eating meat, 36 hours before admission. When three years of age, she had swallowed a solution of lye and, as a result, cicatricial stricture had formed, which had caused lodgment of food on a number of previous occasions. Sometimes a week elapsed before the food passed on.

There had been no attempt at removal on this occasion. Roentgen ray examination by Dr. Manges showed evidence of foreign body in the cervical esophagus. At esophagoscopy, I removed a large bolus of meat in small fragments. No anesthetic or sedative was used. The patient was able to swallow water after removal. The roentgen ray study, after esophagoscopy, showed a stricture of large caliber retaining only the largest size capsule, at the suprasternal notch. The patient showed a four plus blood Wassermann, and was advised to return for dilatation of the cicatricial stricture, and was referred to another department for treatment of her lues.



CASE 2. Foreign body. 1012 T2. *Bolus of Meat in the Esophagus.* A woman, aged 46 years, choked while eating a piece of veal. Following the accident, she had complete aphagia, regurgitating even her own saliva. As a child, the patient had had a lye burn of the esophagus, and on several occasions had had food lodged in the esophagus. Dr. Martha Bailey referred the patient to the Clinic. On admission the patient was in considerable distress, but in good general condition. On esophagoscopy, I found a bolus of meat in the esophagus, just below the cricopharyngeal fold. It was removed, without anesthesia, with forward grasping forceps through a 10 mm. esophagoscope, in 12 minutes and 37 seconds. Following removal, the patient was able to swallow without difficulty. The roentgen ray examination of the esophagus by Dr. W. F. Manges, following removal, showed a very large caliber stricture of the cervical esophagus. No reaction followed esophagoscopy. Patient was discharged well the second day after the removal of the foreign body.

CASE 3. Foreign body. 1057 T8. *Peachstone in the Esophagus.* Negro, male, aged 32 years, four hours before admission, while eating peaches accidently swallowed a peachstone.

Complete aphagia developed; patient was not able to swallow saliva. On admission, the patient was well developed and well nourished, and was suffering very severe discomfort. He was holding his head forward in such a position as to allow the least pressure on the larynx and trachea. There was no dyspnea. The neck showed tenderness on the level of the larynx; there was no emphysema. Roentgen ray examination by Dr. W. F. Manges showed a foreign body, the size and shape of a peachstone, in the esophagus, indenting the posterior wall of the trachea to an appreciable extent. Esophagoscopy was preceded by a hypodermatic administration of the

full physiologic dose of morphin sulphat, one-half grain, with atropin sulphat (grains 1/150) one and one-half hours before operation, with a view to obtaining relaxation because of the size of the foreign body. I exposed the upper esophagus by lifting forward the cricoid with the adult esophageal speculum. The peachstone was tightly impacted just below the cricopharyngeal fold with its base, or stem end, upward. It was grasped with Jackson rotation forceps, alligator type, some difficulty being experienced in securing a firm hold. Gentle firm traction was made, but the foreign body could not be disimpacted by a safe degree of traction. Traction was again made, and the patient was asked to attempt to swallow during traction on the foreign body. As he did so, the cricopharyngeus relaxed, and the peachstone with the esophageal speculum was easily withdrawn.

Following esophagoscopy, the patient complained of considerable soreness in the neck. There was no emphysema, the soreness subsided by the second day, and he was discharged well on the third day following esophagoscopy.

COMMENT: The cooperation of the patient, obtainable because he was not generally anesthetized, was invaluable in

this case; and the usefulness of the voluntary swallowing movements in disimpaction is, I believe, a point of special interest.

CASE 4. Foreign body, 1277 T42. *Peanut kernel in the Esophagus. Cicatricial Stenosis of the Esophagus.* A colored woman, thirty-five years of age, who had cicatricial stenosis of the esophagus as a result of swallowing lye in childhood, while eating peanuts on the day before her admission to the Hospital, noted that a portion of peanut lodged in the esophageal stricture. She had a coughing and gagging attack, and following the accident had complete aphagia. She consulted

Dr. W. F. Kelly, who referred her to the Bronchoscopic Clinic. On admission, her general condition was good. General examination was negative for foreign body, except for regurgitation of everything she attempted to swallow, including saliva. Roentgen ray examination by Dr. Pancoast for opaque foreign body was negative. On esophagoscopy, a portion of peanut kernel was found completely blocking the lumen of the esophageal stricture, at the level of the cricopharyngeal fold. The foreign body was removed with forward grasping forceps. The lumen of the stricture proved to be about 3 mm. in diameter. Following the removal of the foreign body, the patient swallowed sterile water freely. Duration of operation was three minutes and forty-eight seconds. No anesthetic was used. Roentgen ray studies of the esophagus showed cicatricial stenosis, consisting of an upper constriction at the level of the first thoracic vertebra, and a second constriction extending from the fifth to the ninth thoracic vertebrae. The patient, on dismissal the same day, was advised to return for dilatation of her old cicatricial stenosis. This was later carried out.

COMMENT: It is our custom in all foreign body cases in which there is aphagia, to test the function of swallowing before the patient leaves the table by giving him a glass of sterile water to swallow. If it cannot be swallowed, the esophagoscope is passed again to see why. Disease of the esophagus or periesophageal tissues is to be searched for in every case in which food lodges.

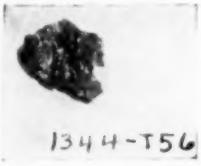
CASE 5. Foreign body. 1286 T43. *Orange Pulp in Esophagus. Compression Stenosis of Hiatal and Abdominal Esophagus, socalled "Cardiospasm."* A boy, 13 years of age, had been referred by Dr. S. Leopold to the Medical Department of the University Hospital, because of marked loss of weight and poor general physical condition, and a history of difficulty in swallowing for a period of 8 years, for study of his condition. Roentgen ray examination by Dr. H. K. Pancoast showed a large dilatation of the thoracic esophagus, and a diagnosis was made of "cardiospasm", socalled. Esophagoscopy was requested by Dr. Chas. Wolferth, of Dr. Stengel's service, to determine the cause of the dilatation of the esophagus.

On esophagoscopy, the thoracic esophagus was found widely dilated, a bolus of orange pulp was found just above the constriction of the hiatus. The orange pulp was removed, and there was found to be a compression stenosis of the hiatus and abdominal esophagus. Duration of esophagoscopy was three minutes and twenty-eight seconds.

COMMENT: This case corroborates the observation of Mosher as to socalled cardiospasm being in some cases an organic stenosis of the abdominal esophagus. While the compressed abdominal esophagus could be dilated to admit a 5 mm. esophagoscope, the compression was so firm as to prevent the passage through of the unmasticated orange pulp. After a few dilatations, the compressed esophagus would permit the passage of a 7 mm. esophagoscope.

CASE 6. Foreign body. 1344 T56. *Bolus of Meat in Cancerous Stricture of the Esophagus.* A man, 48 years of age, who had had marked loss of weight and difficulty in swallowing solid food for seven weeks previous to admission, came to the Clinic for diagnostic esophagoscopy, referred by Dr. P. Brook Bland. Roentgen ray examination by Dr. W. F. Manges showed a filling defect in the dilated portion of the esophagus above the narrowing in its middle third, which suggested the

presence of a foreign body interfering with the proper filling of the diseased area. Esophagoscopy without anesthesia showed a dilatation of the esophagus. A bolus of food was found just above a cancerous stricture, which was located at the hiatal portion of the esophagus, 45 centimeters from the upper teeth. The food was removed through the esophagoscope. A fungating ulcerative lesion was found, from which a



specimen was taken for biopsy. Duration of operation was six minutes and thirty seconds. After removal of the bolus of food, the patient was able to swallow with less difficulty. The histologic examination of the specimen by Dr. B. L. Crawford showed a squamous celled epithelioma.

Case 7. Foreign body. 1101 T14. *Beef Bone in the Esophagus.* Man, aged 44 years, three days before admission, while eating beef soup, choked and felt a sticking sensation in his throat. Roentgen ray examination the next day showed a

foreign body in the cervical region. The patient was then referred by Dr. E. T. Gatewood, to the Bronchoscopic Clinic. On admission, one day later, the patient complained of pain on swallowing, and soreness of the left side of the neck at the level of the suprasternal notch. He had no dyspnea and was able to swallow liquids. Roentgen ray examination, by Dr. W. F. Manges, showed a foreign body at the suprasternal notch. There was slight fever; patient was put on diet of sterile liquids; ice was applied locally to the neck during a period of one week, when all local symptoms had subsided. On esophagoscopy, without anesthesia, I found a spongy piece of beef bone transfixed crosswise, the point perforating the right lateral wall, the other end free and in contact with the left lateral wall, in the esophagus below the cricopharyngeal fold.

The free end of the bone was grasped with side curved forceps and withdrawn with the esophagoscope, allowing the perforating point to trail. This operation required four minutes and thirty seconds. Following esophagoscopy, there was no reaction. The patient swallowed readily, and was kept under observation for two days following removal, and was discharged well.

Case 8. Foreign body, 1125 T17. *Bone in the Esophagus.* Woman, aged twenty-five years, the day before admission, while eating chicken soup, felt something stick in her throat. She inserted her finger, but was unsuccessful in her efforts at

removing the object. Dr. A. Gurin referred the patient to the Clinic. Following the accident, solids were regurgitated, and the patient complained of a pain in her back, which she referred to the right scapular region. She had had no previous difficulty in swallowing. Examination on admission, revealed a well developed and well nourished woman, apparently in great distress. The examination was negative for any signs of foreign body. Roentgen ray study, by Dr. Pancoast, showed lodgment of a capsule in the upper thoracic esophagus. At esophagoscopy, without anesthesia, on day of admission, I found the bone transfixed in the esophagus at the level of the crossing of the left bronchus. The bone was grasped near the free end with rotation forceps and allowed to rotate until the major axis coincided with the long axis of the esophagus. It was then withdrawn in contact with the distal end of the esophagoscope. The operation required two minutes and seven seconds. No reaction followed esophagoscopy, the patient swallowed without difficulty, the pain referred to the back and right shoulder became less severe and was entirely relieved by the second day. The after ray examination showed lodgment of capsule at the end of the first

twenty-four hours, doubtless due to the localized esophagitis in the esophagus, where the bone had been transfixed. The next day a capsule passed without delay. The patient was kept under observation three days and discharged well.

CASE 9. Foreign body. 1132 T18. *Fishbone in the Esophagus.* A young woman, twenty-six years of age, the wife of a physician, felt a sticking sensation in her throat while eating fish (sea bass). She attempted to dislodge the cause of her discomfort with her fingers, but was unsuccessful. She was referred to the Clinic, the day of the accident, by her physician, Dr. Lewis Fisher.

On admission, the patient was very uncomfortable, complaining of pain on swallowing. She was able to take only liquids. The mirror examination of the pharynx was negative for evidence of foreign body. Examination of the neck showed no swelling or emphysema, but marked tenderness was felt on lateral motion of the cricoid cartilage and trachea with the examining finger. The roentgen ray examination by Dr. H. K. Pancoast showed lodgment of the bismuth capsule at the level of the second dorsal vertebra, until the capsule dissolved. On esophagoscopy, without anesthesia, on the day of admission, I found a portion of the vertebra of a fish lodged at the upper thoracic aperture, lying in the coronal plane, a sharp end of bone being slightly embedded in the right lateral wall of the esophagus. The end was freed with the lip of the esophagoscope, grasped with rotation forceps, and pulled into the lumen of the tube. The diameter of the minor axis of the piece of bone was greater than the lumen of the esophagoscope. The esophagoscope was inserted until the bone could be held against the tube and withdrawn with it. Time of esophagoscopy was four minutes and ten seconds. Following esophagoscopy there was no reaction, and the patient was able to swallow with only slight pain. A bismuth capsule, when observed by means of the fluoroscope, hesitated at the former point of lodgment of the foreign body, but was dislodged with the first subsequent swallow of water.

By the second day following esophagoscopy, all soreness had disappeared from the neck, and no hesitation was noted fluoroscopically with the bismuth capsule. Patient was discharged well.

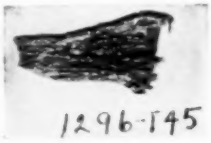
CASE 10. Foreign body. 1290 T44. *Beef bone in the Esophagus.* A woman, 27 years of age, while eating beef soup, felt a sticking sensation in her throat. She inserted her finger and tried to remove the cause of her discomfort, but was unsuccessful. She attempted to eat solid food, but it was regurgitated. She took epsom salts but had no relief. She then consulted Dr. Abraham Epstein, who referred her to the Bronchoscopic Clinic. On admission, her general condition

was good, but she was complaining of severe pains in her neck at the level of the cricoid cartilage. Roentgen ray examination by Dr. H. K. Pancoast showed a shadow suggestive of bone, back of the cricoid cartilage, in the lateral roentgenogram. Lateral displacement of the entire larynx with the examining finger, pressing sidewise at the level of the cricoid cartilage, caused marked increase in tenderness, felt by the patient on the side opposite from the finger. Mirror examination was negative. On esophagoscopy, the larynx was lifted forward by means of the Jackson direct laryngoscope, from behind the cricoid cartilage. The bone was found transfixed in the hypopharynx. It was disimpacted and removed with the alligator forceps. Time of operation was one minute and forty-five seconds. No anesthetic or sedative was used. At roentgen ray examination, after removal, a capsule went down normally. No reaction followed esophagoscopy, and the tenderness in the neck, opposite the side pressed upon, could not be elicited. The patient was discharged well the second day after esophagoscopy.

COMMENT: A valuable diagnostic point that I have never seen mentioned in the literature, and which I often have found very useful, is demonstrated in this case. It consists in the sub-

jective sensation of pain, tenderness, or discomfort noted by the patient, *on the opposite side*, when sidewise pressure is made with the examiner's finger, displacing the entire laryngeal mass laterally. Pressure is to be tried first toward one side, then toward the other. In either case, when present, this sign is diagnostic of foreign body in the hypopharynx or cervical esophagus. It is of value, negatively, only as regards sharp pointed foreign bodies, and it is uncertain when the entire neck is swollen and tender from perforation and infective inflammation.

CASE 11. Foreign body. 1296 T45. *Pork Bone in the Esophagus.* A woman, forty-three years of age, three days before



admission felt a sticking sensation in her throat, while eating a pork chop. No coughing or gagging occurred. She attempted to eat solid food, but it was regurgitated. She was referred to the Bronchoscopic Clinic by Drs. E. P. Becker and E. Dwyer. On admission, her general condition was good. She swallowed liquids with difficulty, and the swallowing efforts caused pain in the neck, referred by the patient to the cricoid level. Local examination revealed some secretion retained in the pyriform sinuses (Jackson's sign of esophageal obstruction), and there was tenderness in the front of the neck at the level of the cricoid cartilage. This tenderness was increased by lateral motion of the trachea and larynx by the examining finger, and was felt on the side opposite the finger. Roentgen ray examination was negative on the films, and a capsule passed readily through the esophagus. The patient was kept under observation for forty-eight hours, it being felt, that as the capsule passed so readily, the discomfort of which the patient complained was probably due to trauma at the former site of lodgment of the foreign body, which had passed on through the esophagus. Ability to swallow improved considerably, but the sign of tenderness and pain elicited by lateral motion of the trachea and larynx persisted. Because of this sign, it was decided to do an esophagoscopy. In introducing the esophagoscope, I found a bone transfixed in the esophagus, just below the crico-

pharyngeus. One point of the bone was deeply imbedded in the right esophageal wall. The point of the bone was disimbedded with the lip of the esophagoscope and the Jackson rotation forceps, alligator type. Traction being made, the long axis of the bone swung around, coming in line with the axis of the esophageal lumen. Because the bone was soft and cancellous, it was then grasped with forward grasping forceps and removed with the esophagoscope. The time of operation was four minutes and twenty seconds. No anesthetic was used. No reaction followed esophagoscopy. The patient swallowed readily following removal of the bone. Local tenderness could not be elicited by lateral motion of the trachea and larynx. Roentgen ray examination was still negative. The patient was discharged well, the second day following esophagoscopy.

COMMENT: This case demonstrates clearly the value of the lateral pressure sign of foreign body in the cervical esophagus, as mentioned in the comment on Case 10.

CASE 12. Foreign body. 1343 T55. *Chicken Bone in the Esophagus.* A woman, forty-five years of age, on the day before admission, while eating chicken soup, felt something lodge in her throat. Following this, she could swallow only liquids, and had the sensation of something sticking in her throat. She consulted Dr. T. C. Davis, who referred her to the Bronchoscopic Clinic.

Her symptoms increased in severity, and she came to the Clinic, the following day. On admission, her general condition was good; general examination was negative for foreign body, except that there was tenderness in the neck at the level of the suprasternal notch. This tenderness was increased by lateral pressure on the trachea. Roentgen ray examination, by Dr. Manges, showed a bismuth capsule lodged just below the cricopharyngeal level, where it remained until it dissolved. On esophagoscopy, a triangular piece of bone was found in the esophageal narrowing at the level of the superior

thoracic aperture. The bone was transfixed, and one point was entering the right lateral wall of the esophagus. The bone was disengaged from the mucosa with rotation forceps, grasped with forward grasping forceps and removed with the esophagoscope. Time, four minutes and forty-five seconds. No anesthetic or sedative was used. After operation, a capsule went down normally. No reaction followed removal of the bone, and the patient was discharged well the next day.

CASE 13. Foreign body. 1353 T62. *Fishbone in the Esophagus. Retropharyngeal Abscess.* A woman, 65 years of age, two days before admission, choked and gagged while eating

fish. Following this, she had a severe pain in her neck and difficulty in swallowing. Dr. G. M. Astley was called, who examined her mouth and throat, but saw no evidence of foreign body. Two days later, she was referred to the Bronchoscopic Clinic. There had been no effort at blind removal by pushing down of the fish bone. On admission, she seemed quite feeble, though very much overweight. She complained of severe pain on swallowing, and of tenderness on the left side of neck, at the level of the cricoid cartilage. The mirror examination of the pharynx was negative, except for a suspicious appearance at the bottom of the left pyriform sinus. Roentgen ray examination was not positive for foreign body, although there was some thickening shown in the tissues posterior to the larynx and cricoid. Temperature on admission was 99.2°. The patient was able to swallow sufficient liquid. The difficulty in swallowing increased, and tenderness in neck persisted. Second roentgen ray study showed no positive evidence of foreign body. Esophagoscopy was done, without anesthesia, and when I lifted the larynx forward with a direct laryngoscope from back of the cricoid cartilage, I found imbedded in the left lateral esophageal wall, below the cricopharyngeal fold, a heavy fishbone. There was

quite a large quantity of pus in the upper esophagus. The pus was aspirated, and the fishbone removed with Mosher forceps. Endoscopy required two minutes, forty-six seconds. The patient's swallowing improved for twenty-four hours. At the end of this time, dysphagia increased. Mirror examination showed marked bulging of the posterior pharyngeal wall, overhanging the larynx. Roentgen ray examination, by Dr. E. P. Pendergrass, confirmed this, and diagnosis of retropharyngeal abscess was made. The abscess was evacuated by placing the patient in Jackson's position for removal of foreign bodies from the pharynx and larynx. The posterior wall was incised after exposure with the direct laryngoscope; pus came up freely and was aspirated from the pharynx and the abscess cavity. Patient breathed much more easily and swallowed with much less difficulty. The abscess cavity was kept open. The temperature came down gradually, and she was discharged ten days later, swallowing normally. The roentgen ray examination on discharge showed only very slight thickening in the retropharyngeal area.

CASE 14. Foreign body. 1068 T10. *Bean in the Right Main Bronchus.* A boy, five years of age, who had been well, three days before admission had a severe choking attack and became cyanotic while playing with freshly shelled beans in his mouth. The attack lasted a half hour, and was followed by wheezing respiration, cough and fever. The second day after the accident, he had a choking attack in which it was thought that he was dying, and a physician was called, who prepared to do a tracheotomy; but the child's condition improved so that tracheotomy was not necessary, and he was sent by Dr. G. H. Deyerle to the Clinic. The wheeze continued, and attacks of dyspnea, coughing, and cyanosis occurred several times.

On physical examination on admission to the hospital, the child appeared well developed and well nourished. He was slightly dyspneic, but not cyanotic, and there was no wheeze

when he was quiet. The chest showed limitation of expansion over the right side, with partial suppression of breath sounds and hyperresonant percussion note over the right middle and lower lobe areas. These physical signs were confirmed by consultation with Dr. Thomas McCrae, and by the roentgen ray examination of Dr. F. F. Bozell, who made a tentative diagnosis, "Right bronchial obstruction, probably due to foreign body."

At bronchoscopy, on the day of admission, I found the larynx and subglottic structures edematous and swollen. Thin whitish pus came up freely. A bean was coughed against the end of the tube, and reaspirated into the right main bronchus. I grasped the bean over the minor axis, with side curved forceps, and brought it against the end of the bronchoscope and withdrew it to the glottis, where, when steady gentle traction was made, the bean broke in the middle, the lower half remaining in the trachea. The upper half, with the greater portion of the hull, was removed with the bronchoscope. I reinserted the bronchoscope, and found the remaining portion of the bean in the right lower lobe bronchus. A fragment of the hull was coughed out through the bronchoscope. Two other fragments of bean were removed with forceps, the smaller through the bronchoscope, and the larger by being held against the end of the bronchoscope. The time of bronchoscopy was fourteen minutes. No anesthesia was used.

The lungs were carefully ausculated, and air was heard to enter all lobes of both lungs freely. Following bronchoscopy, the child's condition improved; dyspnea was relieved. Roentgen ray study showed no obstructive emphysema. Dyspnea, due to subglottic swelling increased, persisted for three days, when it entirely disappeared.

The child was kept under observation for a week. The lungs cleared up, and the child was discharged well.

COMMENT: Beans are perhaps, the most dangerous of all foreign bodies. They are usually, as in this case, aspirated during play, not while eating; hence they are dry and uncooked. Danger arises from two factors: 1. Enlargement of the bean by swelling; and 2. Violent laryngotracheobronchitis from some as yet unknown quality possessed by beans, peas, peanuts, maize, and in fact, most, if not all, vegetable substances.

CASE 15. Foreign body. 1079 T11. *Grain of Corn in Trachea Lodging Alternately in Either Bronchus, in an Asthmatic Child.* Boy, five years of age, was well until seven days before admission, when, while feeding chickens, put a kernel of corn in his mouth, meanwhile laughing and talking to another child. He had a choking attack, and wheezing respiration followed. The child had been subject to attacks of asthma, and the parents thought a recurrence of asthma was the cause of the wheeze. A physician was called, and he treated the boy for asthma for a period of four days; but the wheeze, choking attacks, and dyspnea became worse. The child was

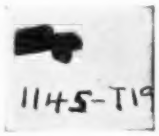
then referred to Dr. Chas. Rebuck, of the Harrisburg Hospital, who referred him at once to the Bronchoscopic Clinic. When he was admitted, he appeared well nourished and well developed. He was slightly dyspneic, but not cyanotic, and made a moaning sound on inspiration. There was slight indrawing at the suprasternal notch. Examination of the chest showed limitation of expansion, hyperresonance on percussion and absence of breath sounds over the entire left chest. The inference was that there was a foreign body in the left main bronchus. Roentgen ray examination the day of admission, by Dr. W. F. Manges, showed the left diaphragm considerably lower than the right, especially at expiration. This indicated partial obstruction to expiration in the left bronchus. The day following admission, distress became more marked, with occasional paroxysms of coughing and dyspnea. The physical signs indicated that a foreign body was loose in the trachea, and a bronchoscopy was done. On insertion of the bronchoscope, it filled with mucopurulent secretion. The grain of corn was coughed against the end of the tube and immediately reaspirated. The lumen of the trachea and bronchi closed completely on cough. The grain of corn was located at the bifurcation, with its tip in the right main bronchus, the large end of the kernel presenting.

I grasped it with light side curved forceps and removed it, using a 5 mm. bronchoscope. The kernel of corn was swollen to double its ordinary size. No anesthetic or sedative was used. Time of operation, 15 minutes. The dyspnea was entirely relieved, but the asthmatic symptoms with wheeze persisted. Temperature was normal. The child was kept under observation for ten days, and at the expiration of that time, the asthmatic attack was relieved and he was discharged well.

COMMENT: The diagnostic value of the asthmatoid wheeze, heard at the open mouth of the patient, as a sign of foreign body in the tracheobronchial tree is well known; but in this case the patient being subject to asthmatic attacks, a less keen diagnostician than Dr. Rebuck might easily have been misled. This is, I believe, the first recorded case of a foreign body wheeze occurring in an asthmatic subject. The persistence of the wheeze after removal of the foreign body would seem to indicate that a genuine attack of asthma had been induced by the presence of the foreign body in the lower air passages. It raises two interesting questions: 1. Is the wheeze in the foreign body cases produced by the same mechanism as the true asthmatic wheeze? and, 2. Is the attack in asthmatic subjects often precipitated by minute unnoticed foreign bodies in the inspired air? For instance, could the nocturnal attacks so common in asthmatics be precipitated at times by the lint of cotton, wool and feathers always to be found in bedrooms?

CASE 16. Foreign body. 1145 T19. *Peanut in Left Main Bronchus Coincident With Beginning Bronchopneumonia.* Boy, 13 months of age, with some prior symptoms of beginning pulmonary trouble, two days before admission to the Bronchoscopic Clinic was eating peanut candy, and had a severe choking and coughing attack. Wheezing sound on breathing persisted, and the child was taken to Dr. D. B. Bliss of Altoona, Pa., who from fluoroscopic studies made a diagnosis of nonopaque foreign body in the left bronchus, and referred the patient to the Bronchoscopic Clinic. On admission, the child appeared very ill. There was slight dyspnea, and the asthmatoid wheeze was present only when the child was active. Chest examination showed lessened expansion over the left lung, especially over the lower lobe. The same area gave hyperresonance on percussion and suppression of breath

sounds on auscultation. The roentgen ray study by Dr. Manges showed obstructive emphysema of the left lung and an accumulation of exudate at the left base. Bronchoscopy the day of admission revealed slight subglottic swelling, and the presence of whitish pus in the trachea. I found a peanut kernel in the left main brochus, just beyond the carina, grasped it and brought it against the end of the tube for withdrawal, but it broke against the tube. A portion of the peanut kernel was coughed out through the tube. Pus came

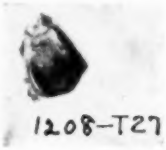


up freely from below the peanut and was aspirated. I reinserted the forceps through the tube, and removed the remaining portion of the peanut. The time of the operation was 7 minutes and forty-five seconds. Following the bronchoscopy, the child's temperature remained elevated, and physical examination showed evidence of inflammatory process in the left lung. Roentgen ray examination confirmed this, and noted, also, the absence of obstructive emphysema, indicating the removal of the foreign body. The patient's temperature gradually came down, and the physical signs cleared. The child was discharged three weeks after admission.

COMMENT: This child, we believe, had a bronchopneumonic process in the left lung, not due to the peanut, but coincident with it, the evidence being that the child had the respiratory infection when the peanut was aspirated. The accurate diagnosis by Dr. Bliss in this case is a confirmation of the accuracy with which a peanut kernel in the lung can be diagnosticated by fluoroscopic examination.

CASE 17. Foreign body. 1208 T27. *Kernel of Corn in the Trachea.* Boy, aged 2 years, was playing with several grains of corn in his mouth when he fell, had a choking attack and became cyanotic. Dr. G. Frank Jones, of Georgetown, Del., saw the child and had a roentgen ray examination made, which was negative; but because of persistent wheezing respiration and cough with general malaise, he brought him to the Clinic, two days after the accident. Examination on ad-

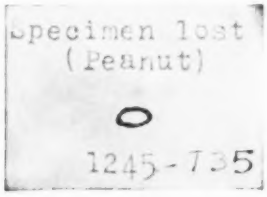
mission showed that the patient was a well developed boy. He was restless, crying, his skin slightly cyanotic, and was quite dyspneic, with a marked "asthmatoid wheeze." There was no retraction at the suprasternal notch, but a distinct tracheal slap and thud on cough could be heard. Examination of the chest showed, on percussion, marked hyperresonance throughout, with bilateral limitation of the chest movement. There seemed to be slightly more evidence of obstruction of



the left than of the right side. Immediate bronchoscopy was decided upon. On passing the bronchoscope, without anesthesia, I found a kernel of corn in the left bronchial orifice, extending into the trachea. Duration of bronchoscopy was two minutes and twenty seconds. Following bronchoscopy, the convalescence was uneventful; very little evidence of subglottic swelling developed, and the child was discharged well on the second day.

Case 18. Foreign body. 1245 T35. *Portion of Peanut Kernel in the Right Lower Lobe Bronchus. Localized Arachidic Bronchitis.* Boy, aged three years, nine days before admission, was eating peanuts when he coughed and gagged. Dr. G. H. Humphreys, of Brockwayville, was called and kept the child under observation for a week, during which the patient had fever and severe cough. Roentgen ray examination the day before admission showed evidence of foreign body in the right lung, and Dr. Humphreys referred the child to the Bronchoscopic Clinic. Examination on admission showed considerable dyspnea and occasionl attacks of short convulsive cough. Roentgen ray examination by Dr. Manges showed extensive density in the right lower lobe practically filling it, which he believed to be exudate rather than atalectasis, because of the lack of displacement of the heart. General examination was unsatisfactory because of the restlessness of the child. Dr. Thomas McCrae reported from his examination, "The signs suggest complete plugging of the lower lobe bronchus on the right side." The child's temperature went up to 101 4/5° the

day of admission, and the morning following was 99°. Bronchoscopy was done, without anesthesia, on the day following admission, and a small portion of peanut was removed from the external division of the right lower lobe bronchus. There was a quantity of pus in the tracheobronchial tree, coming from the right lower lobe bronchus, and I observed that the mucosa of the right lower lobe bronchus was very inflammatory and granular in appearance. Time of bronchoscopy was three minutes and thirty-five seconds. Immediately following bronchoscopy, Dr. McCrae reported improvement in the breath sounds over the right lower lobe. The child's condition improved slightly, but the physical signs of ob-



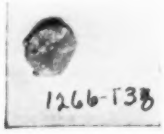
struction in the right lower lobe persisted. Five days after bronchoscopy, Dr. McCrae reported "general condition is better, but there is evidence again of stoppage of the lower lobe bronchus by foreign body or the swelling of the mucous membrane." General condition showed continued improvement, but there was still slight temperature elevation up to 100° at night, and signs of exudate in the right lower lobe in varying amounts at different times. Roentgen ray examination by Dr. Manges at this time showed very little change from the condition found at the first examination. The general condition continued to improve, but there was still evidence of exudate in the right lower lobe on physical examination, and nine days after this examination Dr. Manges reported, "considerable cloudiness in the inner portion of the right lower lobe. No obstructive emphysema. All of the lower lobe functioning except this portion." Diagnostic bronchoscopy was decided upon a fortnight after removal of the portion of peanut. The right lower lobe bronchus was filled with thick, yellowish, odorless pus. Granulations in the lower lobe were removed and pus aspirated. No foreign body was found. Bronchoscopic diagnosis of arachidic bronchitis was made. Temperature rose slightly the first day following bronchoscopy, but came down to 99° following colonic irrigation on the

second day, and did not go above this again. Two days later, roentgen ray examination by Dr. Manges showed thickening along the inner border of the right lower lobe, but improvement over conditions at previous examination. The child returned home one month after admission. There was still some impairment over the right lower lobe, but the temperature had been normal, and the child had been free from symptoms for a period of ten days prior to this time. The patient returned for observation three months after discharge, and Dr. Elmer H. Funk reported that the patient's general condition was good. The child had no respiratory discomfort. There was slight limitation of expansion and impairment of breath sounds over the lower right chest. Roentgen ray examination by Dr. Manges, at this time, showed no evidence of foreign body, only slight peribronchial thickening in the right lower lobe from which the foreign body had been removed.

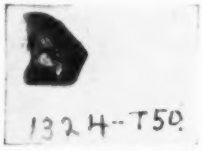
COMMENT: The persistence of signs of bronchial obstruction after removal of peanut kernels is not unusual; and is probably due to obstruction by the mucosal swelling, thick secretions, and sometimes granulation tissue, all of which are the results of the violent reaction set up by peanut kernels, beans, peas, maize, and other vegetable substances, in very young children. The very slow clearing up of the pathology in this case, as compared to others in this series, may have been due to the severity of the inflammatory process induced by the ten days' sojourn of the peanut kernel in the lung; but the possibility of the presence of an additional small fragment of nut kernel in a small branch bronchus keeping up irritation for a time, and afterwards expelled with the copious secretions, must not be overlooked. A child may inhale more than one fragment; or at bronchoscopic removal, a portion of the piece grasped may remain behind.

CASE 19. Foreign body. 1266 T38. *Peanut in the Right Main Bronchus.* A girl, 18 months of age, three days before admission, was eating salted peanuts, when she had a strangling attack. Following this, the child developed a wheeze with occasional attacks of severe paroxysmal cough. The patient had been well before the accident occurred. On admission, when referred to the Bronchoscopic Clinic by Dr. J. B. Rousseau, there was some dyspnea and a marked wheeze on exertion. The chest, which was well formed, showed marked limitation of motion on the right side over the lower

and middle lobe. There was complete absence of breath sounds and marked hyperresonance over the same area. Some air was entering the right upper lobe. Roentgen ray examination by Dr. H. K. Pancoast showed evidence of nonopaque foreign body in the right main bronchus. On bronchoscopy,



without anesthesia, on the second day after admission, I removed a portion of peanut kernel from the right main bronchus, just at the orifice of the right upper lobe bronchus. Duration of operation was two minutes and forty-five seconds. Following the removal of foreign body, air entered freely the lower right and middle lobe. Convalescence was uneventful, and the second day following bronchoscopy, the child was discharged well.



CASE 20. Foreign body. 1324 T50. *Peanut in the Right Lower Lobe Bronchus.* Boy, sixteen years of age, six days before admission was laughing while eating peanuts, and had a choking attack. Some coughing followed, and there was a headache, but otherwise the boy did not feel ill until the evening of the second day. The third day following the accident, Dr. Grala, of Hazelton, Pa., was called and found the boy quite ill with fever and severe cough, which came in paroxysms accompanied by expectoration of blood stained sputum. Roentgen ray examination was positive for nonopaque foreign body in the right lung. Dr. Grala brought the patient to Jefferson Hospital Bronchoscopic Clinic, after much persuasion, the parents feeling that the boy would cough up the peanut. On admission, the boy appeared very ill. There was herpetic eruption on the lips and nares. Slight cyanosis was noted, but no asthmatoid wheeze and no dyspnea. Examination of the chest showed limitation of

motion of the lower half of the right side. There was marked hyperresonance of the percussion note and suppression of breath sounds over the same area, but some air seemed to enter the lower lobe on deep breath. Roentgen ray examination, by Dr. Manges, showed obstructive emphysema and a considerable amount of exudate in the right lower lobe, localizing a nonopaque foreign body in the right stem bronchus. On bronchoscopy, without anesthetic, the second day after admission, I found a peanut kernel in the right lower lobe bronchus, covered with pus and hidden by edematous mucosa. The peanut was visible only during the enlargement of the bronchi on deep inspiration. Pus was aspirated, forceps spaces were determined laterally. Peanut was removed from the right lower lobe bronchus with side curved forceps in six minutes and five seconds. Immediately after bronchoscopy, examination showed air entering all portions of the lower lobe on deep inspiration. Roentgen ray examination, by Dr. Manges, the day after bronchoscopy, showed evidence of exudate still present in the lower lobe, but absence of obstructive emphysema. The temperature, which had been 102° prior to bronchoscopy, became normal the day following, showed a slight rise on the second day, then dropped to normal and remained there. Physical examination showed rapid improvement in the physical signs. By the third day following bronchoscopy, the chest was entirely clear, and the patient was discharged well.

CASE 21. Foreign body. 1331 T51. *Kernel of Corn in the Trachea.* Two days before admission, a boy, three years of age, was playing with a corn in his mouth, and while running and laughing, choked and gagged. Paroxyms of coughing followed. The child was taken to Dr. Charles R. Ogden and Dr. R. J. Nutter, of Clarksburg, W. Va., who referred the child to the Bronchoscopic Clinic. On admission, the child seemed comfortable; there was no dyspnea, no coughing, no wheeze, some evidence of blocking of the right lung, but otherwise, the examination was negative. Roentgen ray by

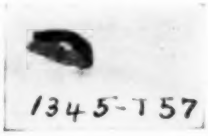
Dr. Leon Solis-Cohen gave evidence of a movable foreign body in the trachea. On performing a bronchoscopy, without anesthesia, on the first day after admission, I found a grain of corn (maize), large end up, projecting into the trachea from the left bronchial orifice. The grain of corn was very much swollen and met with considerable resistance at the glottis. I withdrew it with forward grasping forceps. Time of endoscopy was four minutes and twenty seconds. The convalescence, following removal, was uneventful, and child was discharged well two days later.

CASE 22. Foreign body. 1339 T54. *Kernel of Corn in the Trachea and Left Main Bronchus.* Boy, aged six years, thirteen days before admission, was running with a grain of corn in his mouth, when he choked and strangled. Coughing attacks persisted following the accident. Diagnosis of foreign body in the trachea was made. He was kept under observation, and ten days later was referred to the Bronchoscopic Clinic by Drs. R. E. Mitchell and R. D. Bates. An admission to the receiving ward, the patient seemed tired and sleepy, but gave no evidence of tracheal foreign body. When examined, one hour later, he was found in a sitting posture, and seemed to be trying to suppress a constant desire to cough. A distinct asthmatoid wheeze and an audible slap were heard at the open mouth. He was placed in bed lying on his right side, and after a time the coughing stopped. Examination of his chest in this position gave physical signs of obstruction of the right main bronchus, with a fluttering sound heard over the entire chest. His lips were slightly cyanotic. The upright position in bed was the most comfortable (sitting up); cough and dyspnea increased when he would lie down. The physical signs varied and indicated that the foreign body, which was movable in the trachea, at different times lodged in either bronchus.

On bronchoscopy, I found the larynx and trachea were intensely inflammatory, free pus coming up from below. On

insertion of the bronchoscope through the glottis, the foreign body was coughed against the end of the tube and reaspirated. It was located in the left main bronchus, at the level of the upper lobe bronchus. I removed it with a 5 mm. tube and forward grasping forceps. It broke at the glottis, and a portion was swallowed and afterward recovered in the stool. Duration of operation, one minute and forty-five seconds. A few coarse rales persisted following the bronchoscopy; these cleared entirely by the second day following removal of the foreign body, and the child was discharged well.

COMMENT: The inflammatory condition of the tracheobronchial tree was due partially to the irritating properties and the migratory action of the foreign body, as it cleared up rapidly after removal of the foreign body.



CASE 23. Foreign body. 1345 T57. *Peanut in the Right Main Bronchus.* Boy, aged two and one half years, four days before admission, was given a bag of salted peanuts by his playmates. He choked and gagged for a period of five minutes, and became very red in the face. His mother inverted him and slapped him on the back, and portions of peanut fell from the child's mouth. The child developed wheezing respiration immediately, and had recurrent coughing attacks. Dr. R. Francis Taylor was called three days after the accident, and from his physical examination, made a diagnosis of foreign body in the right bronchus, and referred the child to the Clinic. Examination on admission showed a well developed and well nourished child, having a short, unproductive cough and a pronounced asthmatoid wheeze. Examination of the chest showed limitation in motion with fullness at the right side, marked hyperresonance over the right side, with very small amount of air entering the right lung, as determined by auscultation.

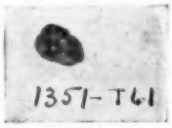
Roentgen ray studies, by Dr. Henry K. Pancoast, confirmed our physical findings of nonopaque foreign body in the right main bronchus, with obstructive emphysema of the right lower and middle lobes. On bronchoscopy, I found a portion

of peanut in the right main bronchus, at the level of the upper lobe bronchus. There was marked inflammatory reaction, with considerable quantity of pus in the right bronchus. The peanut was grasped with forward grasping forceps and removed with four mm. bronchoscope. Time of bronchoscopy was four minutes and twenty-one seconds. After removal, air entered freely all portions of the right lung immediately. The day following bronchoscopy, the physical signs still showed marked obstruction in the right lower and middle bronchi. These signs were noted to be less evident after a severe coughing attack, but on deep inspiration air seemed to enter all portions of the right lung about equally, although with lessened quantity than normal. These findings were confirmed by Dr. Fife, who saw the patient in consultation. Roentgen ray findings confirmed the physical examination, indicating obstruction in the right, middle and lower lobes. I felt that this obstruction was due to swelling of the mucosa and retention of secretion, and not due to unremoved portions of peanut. The physical signs showed only slight improvement, until the fourth day, when there was great improvement. Temperature had not gone above 99.2° and the child's general condition was excellent. By the sixth day, the physical signs of obstructive emphysema had entirely disappeared. Temperature became normal, and the child was discharged well. Examination one month following the bronchoscopy, by Dr. R. Francis Taylor, showed the condition of the child's lung had remained normal.

Case 24. Foreign body. 1351 T61. *Peanut in the Right Lower Lobe Bronchus. Tracheotomy for Laryngeal Dyspnea and Drainage of Lung Before Removal of Foreign Body. Peroral Bronchoscopic Removal.* Ten days before admission to the Bronchoscopic Clinic, the patient, a boy two years of age, while eating peanuts had a severe coughing attack lasting about an hour, becoming very red in the face at times but never cyanotic. Occasional cough and wheezing persisted until admission to the hospital. On the fourth day after the accident, a physician had been consulted, and on examination of the chest, he had stated that there was probably a peanut in the lung. At expiration of another four days, the patient was examined by another physician, and his temperature recorded at 103 degrees. Roentgen ray examinations were made and said to be negative; but because of the progressive increase in the severity of the child's symptoms, the patient was referred to the Bronchoscopic Clinic.

The child was very ill, with a typical peanut toxemia. He had marked asthmatoid wheeze and short unproductive cough. Examination of the chest showed obstructive emphysema of the right lower lobe with a generalized trachebronchitis; abundant free secretion in the trachea. The temperature rose to 106°, but fell to 104° in the afternoon of the same day.

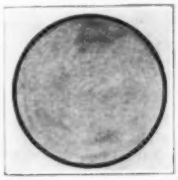
There was considerable dyspnea with retraction at the suprasternal notch; the child seemed to be having great difficulty in getting rid of the secretion in the tracheobronchial tree. It was decided to do a low tracheotomy for relief of dyspnea, and to allow free drainage of the lung. This was



done under local anesthesia; dyspnea was immediately relieved, and large quantities of thick, tenacious pus were aspirated from the tracheobronchial tree with the mechanical aspirator. Thick pus continued to be discharged freely through the tracheotomy tube; the child's general condition showed progressive improvement; the temperature came down to 99° the third day after tracheotomy. The physical signs indicating foreign body in the right lower lobe bronchus persisted. These findings were corroborated by the roentgen ray studies of Dr. Henry K. Pancoast, and it was decided on the fifth day following the tracheotomy to do a bronchoscopy. On insertion of the bronchoscope through the mouth, I found the peanut kernel in the external division of the right lower lobe bronchus, distal to a ring of edematous mucosa. It was visible only on deep inspiration. It was grasped with forward grasping forceps through a 4 mm. bronchoscope and removed. The operation required 15 minutes and five seconds. No anesthetic was used. The child's temperature rose to 100.6°, but came down the same day to normal, and remained there during his convalescence. The right lung cleared rapidly, the tracheotomy wound healed nicely, and on the twenty-first day following the tracheotomy, the child was discharged, the tube having been removed four days previously, as the tracheotomy wound had almost completely healed.

COMMENT: This is a typical case of peanut in the bronchus. The septic bronchitis, the imminent drowning of the patient in his own secretion, the subglottic laryngitis, the physical signs, the roentgen ray signs, the choking and gagging, the asthmatoid wheeze,—all these render the clinical picture complete.

CASE 25. Foreign body. 1015 T3. *Coin (Nickel) in the Esophagus.* Child, female, aged two and one half years, swallowed a nickel while in a moving picture show, one week before admission. She was able to take liquids readily, but solids were regurgitated. She was not seen by a physician



until the day before admission. Roentgen ray examination showed the coin in the cervical region, and she was referred to the Clinic. On admission, the child did not appear ill. Physical examination was negative. Roentgen ray examination, by Dr. Manges, showed the coin in the lateral plane, at the suprasternal notch. At esophagoscopy, without anesthesia, I found the coin in the esophagus just below the cricopharyngeus. The coin was grasped with forward grasping forceps and withdrawn in the lateral plane, in contact with the end of the 7 mm. esophagoscope. Time of operation was two minutes and eleven seconds. No reaction followed esophagoscopy, and the child was discharged well one day after removal of the foreign body.

CASE 26. Foreign body. 1172 T22. *Two Metal Discs in the Esophagus.* Boy, four years of age, five days before admission choked and gagged with two metal discs in his mouth. Castor oil was administered immediately, and when the discs did not pass by bowel, he was taken to Dr. C. A. Copeland, who made a roentgen ray examination, and found the foreign body in the cervical region. Dr. Copeland referred the patient to the Bronchoscopic Clinic. On admission, the child's general condition was good. He was able to swallow solid food. Physical examination was negative. Roentgen ray examination by Dr. H. K. Pancoast confirmed Dr. Copeland's report,

showing a disc shaped metallic foreign body at the level of the suprasternal notch in the lateral plane. On esophagoscopy, without anesthesia, I found the two metal discs, each the size of a twenty-five cent piece, in perfect apposition behind and below the cricopharyngeal fold. The discs were presented with the tip of the 7 mm. esophagoscope, grasped with forward grasping forceps, and removed in the lateral plane in contact with the esophagoscope. No reaction followed esophagoscopy. The patient was kept under observation for two days, and was discharged well.

COMMENT: It might be well to note in connection with this case, that a roentgen ray examination should always be

made after removal of a foreign body, because of the possibility of foreign bodies being multiple. Had it not been known that two discs were present, one of these discs might easily have been removed and one disc allowed to remain in the esophagus, without the knowledge of the esophagoscopist. We make it a rule to have a reexamination by the roentgenologist to show the passages, air or food as the case may be, are free of foreign body, free of any accidental additional foreign body, free from pathology, and that function is fully restored. This completes the record of the case. It is well to remember that serious pathologic narrowing of the esophagus may be the cause of lodgment.

CASE 27. Foreign body. 1241 T34. *Coin (Quarter) in the Esophagus. Removed from the Stomach by Gastroscopy.* A girl, seven years of age, was playing with a quarter in her mouth and swallowed it. She was taken immediately to Dr. E. A. Campbell, who made a roentgen ray examination and found the coin in the lower end of the esophagus. The doctor then referred her to the Bronchoscopic Clinic.

Physical examination was negative for evidence of foreign body. Liquids were swallowed readily. Roentgen ray ex-

amination, by Dr. Pancoast, confirmed the findings of the first roentgenologist. At esophagoscopy, the coin was found just above the level of the diaphragm. On approach with the esophagoscope, it passed through the hiatus, and on following it into the stomach, the edege was presented with the lip of the seven mm. esophagoscope; it was grasped with forward grasping forceps and removed from the stomach. It was

maintained in the lateral plane. Gastroscopy required three minutes and twenty seconds. No anesthetic was used. Recovery was immediate.

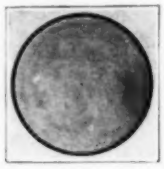
CASE 28. Foreign body. 1257 T37. *Penny in the Esophagus.* Child, one year of age, choked while playing with a penny in her mouth. She was taken to a local physician who told the parents to feed her mashed potatoes and give her castor oil. The penny failed to pass the bowel, and the mother was advised by a visiting nurse to bring the child to the Broncho-

scopic Clinic. On examination, the child appeared normal except for its inability to swallow solid food. Roentgen ray study showed a coin at the suprasternal notch, in the lateral plane. On esophagoscopy, I found the coin, covered with food, lodged at the level of the suprasternal notch, below the cricopharyngeal fold. The food particles were removed by aspiration, the coin presented with the tip of the esophageal speculum, grasped with Mosher forceps and removed in the lateral plane. Time of operation was one minute and thirty seconds. No anesthetic was used. Convalescence was immediate.

CASE 29. Foreign body. 1271 T39. *Penny in the Esophagus.* On the day of admission, the child, a girl, aged five years,

was given a penny with which to play. She immediately put it in her mouth and swallowed it, coughing and gagging. Her mother immediately brought her to the Bronchoscopic Clinic.

On admission, the child seemed well developed and well nourished, and her general condition was good. She complained of difficulty in swallowing solids. Roentgen ray examination, by Dr. Manges, showed a coin shaped foreign body in the esophagus, just below the cricoid. On esophagoscopy,



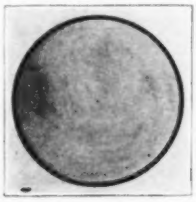
I found the coin at the suprasternal notch. I grasped it with Mathieu forceps and removed it in the lateral plane. Duration of esophagoscopy was three minutes and twenty-seven seconds. No anesthetic was used. The patient was immediately discharged, cured.

CASE 30. Foreign body. 1300 T46. *Coin (Quarter) in the Esophagus.* The morning of the day of admission, a girl, aged

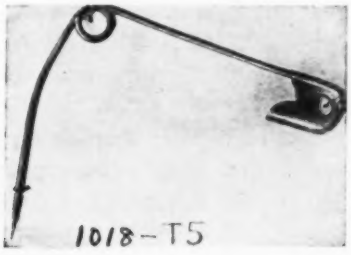
seven years, was lying in bed with a quarter in her mouth, which she swallowed. Pain on attempting to swallow developed immediately, and she came to the receiving ward of the University Hospital. Physical examination was negative. Roentgen ray examination by Dr. H. K. Pancoast showed a coin at the suprasternal notch, in the esophagus, in the lateral plane. A 7 mm. esophagoscope was passed, without anesthesia, general or local. The coin was found just below the cricopharyngeal fold. The folds were manipulated with the lip of the tube mouth until a proper presentation was obtained, with forceps spaces anteriorly and posteriorly. The coin was then grasped flatwise with forward grasping forceps and removed in the frontal plane. Esophagoscopy required two

minutes, three seconds. There was no reaction, and the patient was discharged.

CASE 31. Foreign body. 1348 T59. *Coin (Quarter) in the Esophagus.* Boy, two and one half years of age, was playing with a quarter in his mouth, when it passed downward out of sight in his pharynx. There was no choking or gagging, but he immediately developed inability to swallow solids. On admission, we learned that the child had been a victim of infantile paralysis, and was wearing corrective braces. Roent-

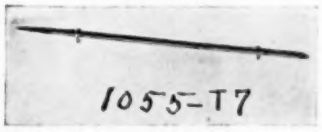


gen ray study, by Dr. Burton, at the Misericordia Hospital, showed the shadow of a coin shaped foreign body at the level of the suprasternal notch. On esophagoscopy, I found the coin just below the cricoid level, posterior to the cricopharyngeal fold. The edge of the coin was presented by means of the esophageal speculum. It was grasped with down jaw alligator forceps and removed with its greatest dimensions in the lateral plane. Immediate recovery.



CASE 32. Foreign body. 1018 T5. *Open Safety Pin in the Esophagus.* Child, colored boy, two and one half years of age, on the morning of the day of his admission to the Hospital, was playing with a number of safety pins. He had a gagging and choking attack, and his mother put her finger down the child's throat, but she was unable to feel the pin. The child was brought to the Bronchoscopic Clinic for examination. He cried on attempt at swallowing, but did not regurgitate liquids. Physical examination was negative. Roentgen ray

examination, by Dr. Pancoast, showed the open safety pin with point up and to the left, keeper toward the right side, with the spring below the cricoid level. The pin was in the cervical region, the expansion being in the coronal plane. On esophagoscopy, I exposed the hypopharynx by lifting forward the cricoid cartilage with the laryngoscope. The keeper of the pin was in the right pyriform sinus, point imbedded in the anterior wall of the left pyriform sinus. The keeper was grasped with rotation forceps, laryngoscope rotated to the left, pin withdrawn through the laryngoscope, retreating the point over its tip. The operation required two minutes, thirty-five seconds. No anesthetic was used. No reaction followed esophagoscopy. The child was discharged well.



Case 33. Foreign body. 1055 T7. *Needle in the Pharynx.* On the day of admission, a woman, 45 years of age, was eating a "hot dog." Taking the first bite, she felt something stick in her throat. She inserted her finger and tried to remove the object, but was unsuccessful. She consulted Dr. A. C. Copeland, who referred her to the Bronchoscopic Clinic. On admission, the patient was complaining of very severe pain at the level of the larynx, which was greatly increased by attempts at swallowing. Mirror examination of the larynx showed the point of the needle embedded in the base of the tongue just above the epiglottis, the shaft extending downward to the bottom of the right pyriform sinus. Roentgen ray examination by Dr. H. K. Pancoast confirmed the location of the needle. The patient was put in the Jackson's position for removal of foreign bodies from the pharynx and larynx; ten drops of a 4% solution of cocain was dropped in right pyriform sinus. The pharynx was exposed with the laryngoscope, the needle was grasped with alligator forceps, the point disimbedded, and the needle removed through the laryngoscope. The operation required one minute and thirty seconds. No reaction followed esophagoscopy, and the patient was discharged well the same day.

CASE 34. Foreign body. 1113 T16. *Open Safety Pin in the Pharynx and Esophagus.* A baby, eight months of age, on the morning of admission was found choking. When the mother examined the child's mouth, she saw a safety pin disappear in the pharynx. The parents were referred to Dr. E. L. Eliason, who had a roentgen ray examination made by Dr. H. K. Pancoast, which showed an open safety pin in the pharynx and esophagus, with the point up. Dr. Eliason referred the child to the Bronchoscopic Clinic.

Examination on admission showed a robust healthy baby, crying lustily. There was no dyspnea, no cyanosis. Liquids were swallowed readily. At specular esophagoscopy, without anesthesia, the safety pin was seen back of the larynx, with the point up and imbedded in the right posterior wall. It was grasped with the Tucker tack forceps, alligator type, and the point disengaged by pushing the pin downward. The point thus protected with the forceps, the pin was removed, without trauma to the tissue. The operation was performed in 55 seconds, without anesthetic. No reaction followed removal, and the child was discharged the following day well.

CASE 35. Foreign body, 1201 T24. *Open Safety Pin in the Hypopharynx.* A baby, seven months of age, was found by her mother crying, gagging and vomiting. The mother missed an open safety pin which she had left sticking in the child's bed, and inferred that the child had swallowed the pin. When

admitted to the Bronchoscopic Clinic, the baby seemed perfectly well, and was contentedly sucking her thumb. Physical examination was negative. Roentgen ray examination by Dr. Cohen showed an open safety pin, point upward and to the right, expanded in the frontal plane in the cervical region, just above the suprasternal notch. On specular esophagoscopy without anesthesia, the hypopharynx was exposed by lifting forward the larynx from behind the cricoid with a Jackson's child's size laryngoscope. The pin was found point upward and toward the right, the spring being below the cricopharyngeus, in the mouth of the esophagus. The keeper was grasped with a Jackson rotation forceps, with which the pin was rotated in the coronal plane over the tip of the laryngoscope, so that the point trailed. It was thus withdrawn along with the laryngoscope without risk of trauma to the tissues. The time of operation was forty-five seconds. The child was discharged well.

Case 36. Foreign body, 1209 T28. *Open Safety Pin in the Esophagus.* On the day before admission, the patient, a baby girl, seven months of age, knocked an open safety pin from her nurses's hand into her open mouth; and before the nurse could recover it, the baby had swallowed it. Dr. Claude L. Holland was called and immediately had a roentgen ray study made, which showed the pin in the child's esophagus. He referred the case immediately to the Bronchoscopic Clinic.

Examination on admission showed a well nourished and fairly well developed child. The patient was on liquid diet and took its nourishment without difficulty. Roentgen ray examination by Dr. Manges showed a small open safety pin, point down and to the left, keeper to the right, at the cricopharyngeal region in the esophagus. Forward displacement of the trachea indicated considerable inflammation of the posterior esophageal wall. At esophagoscopy, I found the pin in the esophagus below the cricopharyngeal fold, the

point downward and to the left, imbedded in the lateral wall of the esophagus. The foreign body was presented with the esophageal speculum, the spring end was grasped with Mathieu forceps, and was removed through the esophageal speculum. No anesthetic was used. Time of operation was two minutes and twenty-five seconds. There was no reaction following esophagoscopy. The child was discharged well the second day after removal.

COMMENT: The danger of overriding and pushing downward a safety pin lodged point downward should be emphasized. Fatal puncture might easily result from overlooking this possibility.

CASE 37. Foreign body, 1217 T31. *Common Pin in the Pharynx.* Young lady, 17 years of age, had two pins in her mouth, and discovered that one had been swallowed. Examination was made, and the patient was then referred to the Clinic. Mirror examination showed the pin in the hypopharynx, to the right of the epiglottis, point up. Roentgen ray examination by Dr. H. K. Pancoast confirmed the location and direction of the pin. Ten drops of 4% solution of cocain were dropped into the pharynx, with the patient dorsally recumbent. The patient was placed in the Jackson position for the removal of foreign bodies from the larynx and hypopharynx. The hypopharynx was exposed with the direct laryngoscope, the point of the pin was grasped with a Jackson forceps, and removed through the laryngoscope in one minute and ten seconds. No reaction followed removal.

CASE 38. Foreign body, 1306 T48. *Open Safety Pin in the Hypopharynx.* A boy, two years of age, on the day before his admission, picked up a safety pin from the bed beside him, where the mother had put it while she was changing his clothing, put it in his mouth, and swallowed it. He choked and

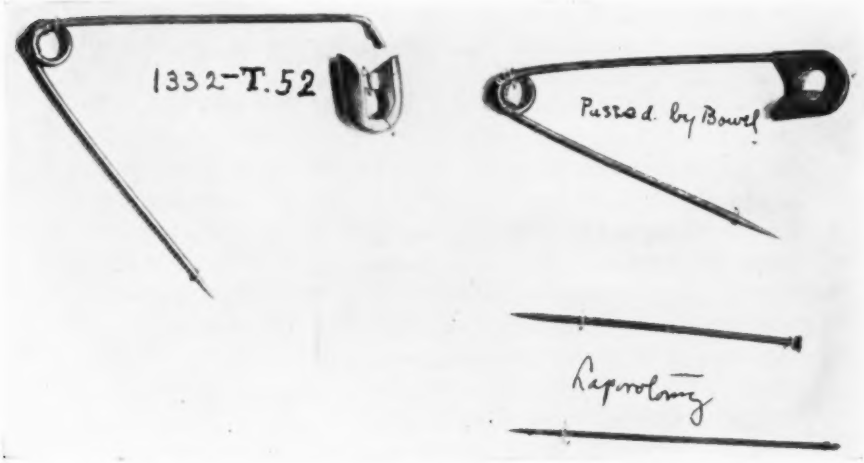
gagged, and the mother's efforts at removal with her finger, were unsuccessful. The child was taken immediately to Dr. John Atlee, of Lancaster, Pa., who had a roentgen ray study made, and found the pin in the esophagus. He referred the patient to the Bronchoscopic Clinic. On esophagoscopy, without anesthesia, I found the pin open, point up and to the right, spring below the level of the cricopharyngeus. The

direct laryngoscope and Jackson alligator forceps were used to withdraw the pin safely, rotating it in the frontal plane by grasping the keeper. The time of the procedure was one minute and forty-seven seconds. No anesthetic or sedative was used. No reaction followed esophagoscopy. The child was discharged well the second day after removal.

Case 39. Foreign body, 1310 T49. *Safety Pin in the Esophagus.* A six months old baby girl, on the day of admission, while her mother was dressing her, took a pin which her mother had placed beside her on the pillow, put it in her mouth and swallowed it. She choked and gagged. An aunt took her by the heels and slapped her on the back. The child became cyanotic, and the aunt put her finger in the baby's mouth and pushed the pin down so that the child could breathe. The baby was taken to Dr. Ralph Hollingshead, of Westville, N. J., who referred the case to the Cooper Hospital. At the Cooper Hospital, a roentgen ray study showed the pin in the lower cervical region. The child was referred to the

Bronchoscopic Clinic, no attempt at removal having been made.

On admission, the child was comfortable and nothing abnormal was noted. Roentgen ray examination by Dr. Manges confirmed that made at the Cooper Hospital. On esophagoscopy, without anesthesia, I found the pin in the hypopharynx, open, the point imbedded in the left pyriform sinus, and the spring below the cricopharyngeal fold. The imbedded point was disengaged with the Tucker pin forceps, alligator type. The pin was then rotated in the sagital plane and withdrawn, with the point trailing to avoid trauma. Time of operation, 5 minutes, 2 seconds. No reaction followed removal; child was discharged well at the end of twenty-four hours.



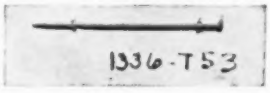
Case 40. Foreign body, 1332 T52. *Safety Pin in the Esophagus. Multiple Foreign Bodies in the Alimentary Canal.* A woman, 36 years of age, an inmate of a state hospital for the insane, obtained a number of pins and needles and swallowed them. After the occurrence, she gagged, choked, and complained of a sticking sensation in her pharynx. There was considerable difficulty in swallowing, although she was able to take sufficient liquids. There were no symptoms except the sticking sensation in the right side of the neck on swallowing. Physical examination showed the patient in good general condition. Mirror examination of the pharynx showed a small portion of the keeper of a safety pin in the left pyriform sinus.

Pressure on the right side of the neck elicited tenderness. Otherwise the examination was negative. Roentgen ray examination showed a large open safety pin in the hypopharynx, the lower portion being in the upper esophagus; there was another open safety pin, a common pin, and a needle in the upper abdomen. The pharynx was exposed with the direct laryngoscope, the point of the pin, which was toward the left, was found imbedded deeply in the left lateral wall of the pharynx. The pin was very large and quite stiff. The hypopharynx was exposed, the pin was grasped with rotation forceps, and rotated over the tip of the laryngoscope toward the left and withdrawn, while making pressure toward the right lateral wall, with the Jackson alligator forceps. The dysphagia disappeared after removal of the pin. It was decided to observe the progress of the intestinal foreign bodies by means of the fluoroscope from day to day. These observations showed the pins moving slowly through the gastrointestinal tract. The safety pin passed by bowel two weeks after the removal of the other safety pin from the pharynx. The pin and the needle were both in the right lower abdomen, and did not seem to be moving. Daily observations, for a period of six days, showed no change of positions. A consultation was held with Dr. Thomas A. Shallow and Dr. Chevalier Jackson. It was deemed dangerous to wait, lest the points perforate by ulceration, because of the unchanged position. Laporotomy was done by Dr. Shallow. The needle and pin were found in the transverse colon and removed. No reaction followed the operation, and the roentgen ray examination by Dr. Manges showed absence of any radiopaque foreign body anywhere in the air and food passages. The patient's physical condition was good, although mentally she was quite melancholic. She was kept under observation two weeks following the operation. The wound being healed, she was discharged in good general condition. A final ray examination was made before discharge. This showed the absence of opaque foreign bodies from the gastrointestinal tract.

COMMENT: This case illustrates the point always taught by Chevalier Jackson, as to the necessity of ray examination from the nasopharynx to the tuberosities of the ischia, in order to discover (or exclude) the presence of bodies in the air and food passages. Examination of the esophagus only in this case would have missed the intestinal foreign bodies, and

these would, in this instance, have probably proven fatal. It is interesting to note that one open safety pin passed through, while a needle and a pin lodged. It seems well also to note the wisdom of a final ray before the discharge, advisable in any case, but especially so in cases of insane or hysterical patients.

CASE 41. Foreign body, 1336 T53. *Common Pin in the Right Stem Bronchus.* A baby girl, nine months of age, while playing on the floor, suddenly had a violent gagging and vomiting attack. The mother inserted her finger in the child's mouth and felt a sharp object which she thought was a pin, but she was not able to remove it. Dr. Matthew Ersner was called and had a roentgen ray examination made by Dr. George Pfhaler, which showed a common pin in the right stem bronchus. The child was then referred by Dr. Ersner to the Bronchoscopic Clinic.



Examination on admission, the day of accident, showed a baby who seemed perfectly well, with slight cough, but no localizing signs of foreign body. On bronchoscopy, I found the larynx slightly inflammatory, and a 4 mm. bronchoscope fitted very closely in the subglottic space. The mucosa was pale, but otherwise normal. A thread like line of pus was found on the posterior tracheal wall, leading into the right bronchus. The bronchoscope was brought in line with the right bronchus and advanced to the level of the upper lobe bronchus, when the child coughed violently, and the pin was projected through the bronchoscope, striking the right lens of my glasses. The duration of the operation was one minute, ten seconds. No anesthetic was used. Following bronchoscopy, the child was quite dyspneic, but was getting sufficient air. The second night following the bronchoscopy, dyspnea became so great that tracheotomy seemed imminent, but it gradually subsided, and on the fourth day, all evidence of subglottic swelling had disappeared. The temperature had not been above 100, and as dyspnea subsided it came to normal. The child was discharged well the fourth day after bronchoscopy.

CASE 42. Foreign body, 1368 T69. *Safety Pin in the Esophagus.* A child, two years of age, while her mother was dressing her, picked up an open safety pin, put it in her mouth, and choked and gagged. Mother slapped the child on the back and attempted to remove the pin from the throat with her finger. Her efforts were unsuccessful and she took the child immediately to the Misericordia Hospital, where roentgen ray examination showed the safety pin in the esophagus, open, with the point up, at the level of the suprasternal notch. The following day, the child was referred to the Bronchoscopic Clinic. On admission, the child's general condition was good.

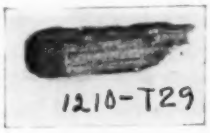
She swallowed liquids readily, but her mother stated that solid food was regurgitated. There was no tenderness or emphysema of the neck. Roentgen ray examination by Dr. E. P. Pendergrass confirmed the finding of the examination made at the Misericordia Hospital. On esophagoscopy, the pin was found at the level of the suprasternal notch, the keeper to the left, the pin open, point up and to the right. The pin was open at an angle of 45 degrees. It was rotated in the coronal plane by grasping the keeper with rotation forceps, and was removed with the point trailing, in order to prevent trauma to the mucosa of the esophagus. Time of operation, one minute and 32 seconds. No anesthesia was used. No reaction followed esophagoscopy. The child was kept under observation for a period of three days. Its condition remained normal, and at the end of this time it was discharged, well.

CASE 43. Foreign body, 1091 T12. *Button in the Esophagus.* A boy, 14 months of age, eighteen days before admission, swallowed a button. Following the accident, the child was able to swallow liquids readily, but solids were regurgitated.

One week later, he was taken to a physician, who made an examination and referred him to the Bronchoscopic Clinic.

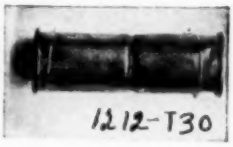
On admission, the child showed no reaction after twenty-four hours observation. Roentgen ray examination, by Dr. Manges, showed a button in the lateral plane, at the level of the suprasternal notch. The patient's general condition was good. On esophagoscopy, using a 7 mm. esophagoscope, I

found the button just below the cricopharyngeal fold, grasped it with forward grasping forceps, removing it in contact with the esophagoscopic tube mouth, maintaining the greater dimension of the button in the lateral plane. Removal required four minutes and forty seconds. There was no reaction following removal of the foreign body. The child was discharged the second day, well.



CASE 44. Foreign body. 1210 T29. *Splinter of Wood and Pieces of Dirt in the Trachea.* A girl, thirteen months of age, who was an inmate of an almshouse for feeble minded children, was found strangling and coughing when another child had forced a handful of dirt and pebbles into her mouth. Up to this time the baby had been well, though she had a congenital double harelip and entire absence of the palate. The foreign material was removed from the child's mouth and throat, but she was still so dyspneic, that the possible necessity of tracheotomy was considered. Roentgen ray examination gave evidence of foreign body in the trachea, and the child was referred by Dr. Gillette to the Clinic. On admission, the second day of illness, the child was extremely ill, very restless, slightly cyanotic, with rattling sound on inspiration, but no indrawing at the suprasternal notch. The chest

was emphysematous in type, hyperresonant throughout, with an element of tympany over the left lung, with restriction of motion over the left chest. After fluoroscopic study, Dr. Manges gave a positive diagnosis of a nonopaque tracheal foreign body. Bronchoscopy was done without anesthesia, and a splinter of wood removed from the trachea. Considerable dirt and foul pus were bronchoscopically aspirated from the trachea and bronchi. Breathing improved following the bronchoscopy for several hours, but then became very rapid and cyanosis increased. Tracheotomy was done with the thought of facilitating drainage, though there was very little evidence of laryngeal dyspnea. The secretion was aspirated, but the patient's condition became progressively worse, and the child died within twelve hours from the diffuse septic bronchitis.



CASE 45. Foreign body, 1212 T30. *Pencil Cap in the Right Main Bronchus.* One day before admission, a girl, aged 11 years, was lying in bed with a pencil cap in her mouth, and on laughing it disappeared. Dr. W. B. Glendon, of Bridgeton, N. J., was immediately called. Roentgen ray examination showed the pencil cap in the right bronchus, and Dr. Glendon referred the child to the Bronchoscopic Clinic. Wheezing had persisted, following the accident, and pain was felt in the right side of the chest. On admission, the child was in good condition, had a slight cough and slight wheeze on exertion. Examination of the chest showed some limitation of motion on the right. Percussion was resonant throughout both lungs. Breath sounds were slightly harsh on the right side; air passed in and out freely from the right lung. On bronchoscopy, without anesthesia, the day following admission, I found the pencil cap in the right bronchus, and removed it by grasping the upper edge with side curved forceps and covering the lower edge with the lip of the tube. Time of bronchoscopy was two minutes and fifty-five seconds. Following removal, recovery was uneventful. Nothing abnormal could be detected by physical examination in either

lung. The child's temperature rose slightly, but following administration of a laxative and its free action, it became normal. Child was discharged well the second day after bronchoscopy.

CASE 46. Foreign body, 1235 T33. *Metal Key in the Esophagus.* Boy, two years of age, while playing on the floor, choked and gagged. A small skate key or radiator air valve key was missed, and the child was supposed to have swallowed it. He coughed and vomited, but the foreign body was

not regurgitated. The child was able to swallow only liquids thereafter.

When the patient was admitted to the Bronchoscopic Clinic, his general condition was good. Examination for foreign body was negative, except for the fact, that while being examined, he had a coughing attack and regurgitated some food. Roentgen ray examination showed a key at the level of the suprasternal notch. On esophagoscopy, a metal key was found just below the cricopharyngeus in the cervical esophagus. It was lying with its long axis in the lateral plane, handle up. On inspiration, it would disappear behind the cricopharyngeal fold. It was presented with the lip of the esophagoscope, grasped with rotation forceps through the hole in the handle, and withdrawn in the lateral plane, allowing time for the relaxation of the cricopharyngeus, which grasped it firmly on attempt at withdrawal. Time of operation was six minutes and fifteen seconds. No anesthetic or sedative used. Recovery was immediate. The child was discharged, but returned the following day for observation, and was then found to be perfectly well.

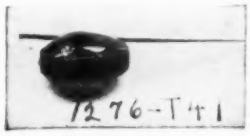
CASE 47. Foreign body, 1275 T40. *Skirt Weight in the Esophagus.* Two weeks before admission, a girl, three years of age, was playing with a lead skirt weight in her mouth; she choked and gagged, and became cyanotic. She was unable

to swallow anything but liquids following the accident and the next day she was taken to a physician, who told them to wait, that the dress weight would pass by bowel. The parents waited two weeks and then took the child again to the physician, who referred the case to the Bronchoscopic Clinic. On admission, the child's general condition was fair; she was fairly well nourished, but her skin was pale, and she complained of soreness and stiffness in her neck. There were no tracheobronchial symptoms. Examination was negative except for tenderness in the neck over the trachea, at the level of the suprasternal notch. Roentgen ray examination revealed a disc shaped foreign body at the suprasternal notch, its greater

dimension in the frontal plane. On esophagoscopy, I found the cervical esophagus filled with foul smelling pus, the mucosa was granular and intensely inflammatory. The lead skirt weight was found at the level of the suprasternal notch, covered with food debris. The edge of the foreign body was presented with the lip of the esophagoscope (6 mm.) grasped with forward grasping forceps, and removed with its greater dimension in the frontal plane. The time of the operation was thirteen minutes and thirty seconds; no anesthetic or sedative was used. There was no reaction following esophagoscopy; the child swallowed liquids and soft foods readily on the first day after the operation. On the second day following removal of the foreign body, her general condition was good, and she was discharged well.

Case 48. Foreign body, 1276 T41. *Glass Bead in the Right Stem Bronchus.* Girl, aged four years, on the day of her admission to the Hospital was playing with a string of beads, when she came to her mother saying she had choked on one. She was taken by the heels and inverted, and immediately became very dyspneic. She was then taken to the office of Dr. Wm. Quicksall, who succeeded in relieving her dyspnea by pushing the bead from her larynx into the trachea with his finger. He

then brought the child to the Clinic at the University Hospital for removal of foreign body. Examination showed a well developed and nourished child, quite relaxed and exhausted in appearance. There was no cough, but a distinct asthmatoid wheeze was heard at the open mouth. On voluntary cough, the audible slap and palpatory thud of tracheal diagnostic foreign body were present. The chest was well formed, but showed limited expansion on the right. Breath sounds were very much lessened over this area, with hyperresonant percussion note. The wheeze was heard loudest over the right bronchus posteriorly. Roentgen ray examination by Dr. H.



K. Pancoast showed an elongated glass bead in the right stem bronchus. Bronchoscopy was done without anesthesia on the day of admission. On insertion of the bronchoscope, the bead was coughed against the end of the tube, and was aspirated into the left lower lobe bronchus. From this location, I removed it with deeply cupped forward grasping forceps. Duration of operation, fourteen minutes and twenty-nine seconds. Convalescence was uneventful; physical and roentgen ray examinations were negative on the second day after bronchoscopy, and the child was discharged well.

COMMENT: Inversion of the patient was done by the parents in this case, and is a dangerous procedure because of the risk of impaction to the foreign body in the glottis.

CASE 49. Foreign body, 1346 T58. *Drain in Pleural Cavity.* A young woman, twenty-two years of age, had a thoracotomy for empyema. While she was being dressed by her surgeon, a rubber drainage tube slipped into the pleural cavity, and could not be recovered by means of dressing forceps. Pleuroscopy was done, without anesthesia, by insertion of a 5 mm. pleuroscope, through the thoracotomy wound.

I found the tube just within the outer wall of the cavity, on the upper surface of the diaphragm. I grasped it with forward grasping forceps and removed it.

Duration of operation was one minute and thirty-eight seconds.

Case 50. Foreign body, 1355 T63. *Bullet in the Right Main Bronchus (Aspirated).* Boy, aged five years, four days before admission to the Bronchoscopic Clinic, was playing with what was supposed to be a marble, in his mouth, and while laughing aspirated it. Severe coughing and choking attacks followed. A local physician, after a roentgen ray examination, determined that the foreign body was in the right bronchus. The patient was afterward referred to the Bronchoscopic Clinic.

Examination on admission showed that the child was well developed and well nourished, not dyspneic, hoarse, or croupy. The physical examination revealed partial obstruction in the right main bronchus. At this time, there was no evidence of tracheal foreign body. Later, the same day, the child was observed after a severe coughing attack, in which he had become cyanotic, and a definite auditory slap and palpable thud were elicited. The foreign body was reaspirated into the right main bonchus. It was determined to do a bronchoscopy without delay, because of the danger of asphyxiation. The roentgen ray examination by Dr. Manges confirmed the physical findings, but it was noted that the foreign body was

very dense and had one surface slightly flattened. At bronchoscopy, without anesthesia, I found the foreign body in the right main bronchus, at the orifice of the right upper lobe bronchus. It was the color of a toy marble as seen through the bronchoscope, but showed two flattened surfaces. It was grasped with globular object forceps such as we use for marbles, but it was found that they would not hold. It was then grasped with deeply cupped forward grasping forceps, which held securely, and the foreign body was removed. After removal, it was found to be a lead bullet. Time of endoscopy was thirteen minutes and thirty-three seconds. The air entered freely all portions of both lungs after bronchoscopy, and no reaction followed.

A few rales persisted in the right side of the chest up to the second day after bronchoscopy. The patient was discharged, well, the second day following removal of the bullet.

Summary.

Esophageal Foreign Bodies	33
Removed by Esophagoscopy or Gastroscopy	33
Removed by Esophagotomy	none
Patients recovered	33
Patients died	none
Tracheobronchial Foreign Bodies	17
Removed by peroral Bronchoscopy	17
Coughed up before second Bronchoscopy	none
Patients recovered	16
Patients died	1
Total number foreign bodies in which Endoscopy was done	50
Total removed (peroral)	50
Total recoveries	49
Total Endoscopic mortality	none
Total ultimate mortality	1

One case of splinter of wood and dirt in tracheobronchial tree; the child was moribund on admission, and died of septic bronchitis within 12 hours of admission.

Conclusions.

From observations in this series of cases, the following points seem especially worthy of mention.

1. "An ounce of prevention is worth a pound of cure," and foreign body accidents are nearly all preventable.

2. The great majority of foreign body cases are children, victims of the carelessness of those in charge of them.

3. It is a matter of vital importance to exclude foreign body in all acute and chronic diseases of the larynx and lungs. Hoarseness and increasing dyspnea due to foreign body in the larynx may simulate diphtheria; drowned lung due to complete blocking of a bronchus by foreign body has been thoracotomized for "empyema," with negative results. (Subsequent bronchoscopic removal of foreign body cured the patient.)

4. Foreign body should be excluded in all cases of the slightest abnormality in swallowing. The roentgenologic determination of the swallowing function is of the greatest importance in this connection.

5. It is the general practitioner on whom this responsibility rests. How well he has measured up to this requirement is shown by the fact, that in fully 90 per cent of the cases, the diagnosis was correctly made by symptoms, physical signs, and roentgen ray studies, before the patient was admitted to the Bronchoscopic Clinic.

6. Foreign body does not ordinarily require emergency operation. However, cases do occur that call for immediate relief. *Urgent dyspnea,* with indrawing at the suprasternal notch demands tracheotomy to save the patient's life, *not,* however, for the purpose of *lower bronchoscopy. Aphagia, with symptoms of water starvation,* demands gastrostomy, with hypodermoclysis, enteroclysis, etc., as temporary measures, to supply the urgent demand for water and food. The emergency being over, careful study of the case may be carried out and orderly removal done where adequate instrumentarium and properly trained bronchoscopists are available.

7. Urgency of removal depends on the character of the foreign body, its point of lodgment, and the extent and rapidity of progress of the pathologic process due to the presence of the foreign body. Vegetable substances, because of their irritating properties should be removed as soon as the foreign body is definitely localized and the proper bronchoscopic technic is available. Beans, peas, maize, and peanuts increase greatly in size when subjected to moisture and warmth, and when they occur as tracheal foreign bodies, they are likely to produce asphyxia because of this swelling and the inflammatory reaction in the trachea which their presence excites.

They should be removed early. Drowned lung or atelectasis due to the presence of foreign body requires early bronchoscopic removal of the foreign body for drainage.

8. The desirability of peroral bronchoscopic removal without general anesthesia is demonstrated. The use of no anesthesia, or in adults local anesthesia only, permits the cooperation of the patient and guards against the danger of respiratory arrest. General anesthesia is very dangerous in dyspneic patients. Morphin may be used as a sedative in patients free from dyspnea.

9. The necessity for teamwork and the importance of the correct position of the patient is amply demonstrated. Without these two elements, failure would have been inevitable in a number of cases.

10. The difficulties in the solution of the mechanical problems of removal in the individual case require special study. After adequate instrumentarium and proper bronchoscopic technic is acquired, the working out of the problem on the rubber tube, dog and cadaver is undoubtedly the best preparation.

11. The percentage of morbidity and mortality in the endoscopic procedures per se in this series of cases is nil.

REFERENCES.

1. Jackson, Chevalier. (a) Peroral Endoscopy and Laryngeal Surgery, Text Book. Laryngoscope Co., 1914. (b) Observations on the Pathology of Foreign Bodies in the Air and Food Passages. Mutter Lecture, 1917. Surg. Gyn. and Obstet., Mar., 1919. (c) Brochoscopy and Esophagoscopy. W. B. Saunders Co., 1922.
2. McCrae, Thomas. Clinical Features of Foreign Bodies in the Air Passages. Lumlein Lectures, 1924. Lancet.
3. Manges, Willis F. Atelectasis as a Roentgen Ray Sign of Foreign Body in the Air Passages. American Journal of Roentgenology and Radium Therapy, vol. II, No. 6, June, 1924.

THE PRESENT STATUS OF ELECTROTHERAPEUTIC MEASURES USED IN THE PRACTICE OF OTO-LARYNGOLOGY.

JOSEPH C. BECK, M.D. AND HARRY L. POLLOCK, M.D.

CHICAGO.

In recent years, many articles have appeared in our leading journals dealing with subjects closely allied with that which we are treating in our presentation today. Naturally, most authors present an optimistic outlook, because they usually give their results with one and only one of the various methods under discussion today, as for instance, diathermia, ultraviolet rays, X-ray, etc. Others have followed their methods, but apparently do not obtain satisfactory results, and early discard these procedures, stating that they cannot procure the good results outlined by the original author. Most of these failures are easily understood by us.

While we are all primarily oto-laryngologists, very few of us have studied the physics of electricity, or really know anything of the physiologic effects of the various currents used. We do not know that a slightly larger dose of some currents will have an opposite effect from a smaller dose. In the larger hospitals and clinics, where there is a medically trained electrotherapeutist in charge of the various appliances, one can expect better results than where individual oto-laryngologists must do their own treating. For example, a new electric appliance may have been used successfully in a large clinic, and the results published in one of our well recognized journals. Immediately there is a demand for such appliance from various physicians throughout the country. They know very little of the forms of electricity being used, and the information as to its use is so limited, that they are compelled to work out their own method, which, due to a lack of knowledge of the essentials of electrotherapeutics, often ends in poor results. Another cause of failure lies in the fact, that the manufacturers of these various devices usually claim highly successful results and cures in many pathologic condi-

tions from their particular apparatus in order to obtain a sale. Frequently, claims are made which we know to be impossible. Most physicians, having bought the apparatus, often at a considerable expense, are tempted to treat all sorts and varieties of conditions, so as to reimburse themselves partially for the original outlay with which they have burdened themselves.

At present, we might say that we are living in an electrical age, as nearly all the modern improvements of our daily life are due, in one form or another, to electricity. So, too, has the electrotherapeutic advancement been made, and each day brings forth something new in this field.

Some of the older currents, which have been used for years, are well known to all of us, as is their limited application. It is not the purpose of this paper to delve deeply into the history of the various forms of electricity which have been or now are being used, as that phase would take up all the time at our disposal. The form of electricity that was used most extensively in early modern medicine is the galvanic and faradic currents. The galvanic, the direct or continuous current, is a low tension one. Its use in living tissues is always productive of marked polarity effects in the tissues immediately adjacent to the electrodes. The faradic is the alternating or broken current.

The principal uses of these two currents are: (1) to stimulate muscles, (A) those in which the controlling nerve has been destroyed, and (B) those atrophied from disuse; (2) to test muscles for reaction of degeneration. To refresh our memory, the following results occur in testing muscles: If a galvanic current, with one electrode applied to the muscle, the other at any indefinite point, is suddenly switched on or off, muscular contraction occurs. The negative, or kathode, is the most active in closing the current (KKC>ACC). The positive, or anode, is the most active in opening the current (AOC>KOC). The nutrition and tone of a muscle fiber is governed by nervous impulses originating in the motor nerve cells situated in the anterior horn of the spinal cord, and if these are cut off, the muscle undergoes degeneration. This reaction of degeneration was first described by Erb in 1868. In testing, we find that there is absolute loss of all response to stimulation by the faradic current because faradism stimulates only the nerve. The muscles respond to interrupted galvanism by a slow, sluggish and long continued contraction,

which passes off gradually. In normal muscle, the Kathodal Closing Contraction is obtained more readily than the Anodal Closing Contraction. In most cases of degenerated muscles this is reversed, and the ACC is obtained first. If the Anode C. C. gives a response with a lesser current than the Kathode, diagnosis of the reaction to degeneration is made. The use of the galvanic, in our hands, has been exceedingly useful in determining this reaction of degeneration, and, secondly, in stimulating the muscles to prevent them from degenerating. This is especially true of the muscles supplied by the facial nerve, whether the nerve has been destroyed by surgical procedure, by lues, or by a peripheral neuritis, such as Bell's palsy. The faradic is of use in stimulating the nerve only as stated before; it has no action on the muscle itself.

A Sinusoidal current is a current, the voltage tracing (oscillograph) of which resembles a sine or segment of a circle. The rapid sinusoidal current is the ordinary commercial alternating current, and voltage tracing of it would show as many positive and negative sines as there are cycles per second. A cycle is one complete positive and negative phase. A slow galvanic sinusoidal current is the straight galvanic current modified by being run thru a constantly and evenly varying resistance, which is so constructed that each succeeding wave is of the opposite polarity to that of the preceding one. We employ the rapid sinusoidal current in stimulating relaxed or atonic muscles, where no reaction of degeneration has occurred. Where this has occurred, we employ the slow sinusoidal, because of fear of overstimulation which would weaken the muscle more and more, and thus our end results would be disastrous. Thus, it can be seen that the theory of physics must be understood in order to obtain the best results.

High Frequency. This is a current having a frequency of oscillation measured in the hundreds of thousands or million a second, usually anything that is above thirty thousand oscillations per second, i.e., having such a high rate of alternations, that living tissues do not attempt to contract under each impulse (Sampson). There are two forms employed in medicine, the general and the local. The general is never employed in oto-laryngology, except if we desire a general metabolic action. General high frequency (autocondensation) gives the following results: Increased activity of meta-

bolism and lower blood pressure. In local application of high frequency, sweat and mucous glands are stimulated. Moderate vasodilitation occurs, and local hyperemia results. The results are that painful conditions are eased and inflammatory exudates resolved (J. Magnus Redding). The local high frequency is applied to any part of the body, but in our hands, we have not seen any benefits (except perhaps the psychic), and therefore do not recommend its use.

Leucodescent Heat and Diathermia. According to Sampson, there are three kinds of heat employed as therapeutic measures: (1) Conductive, (2) Convective, (3) Conversive. *Conductive* heat is heat applied to the body by continuity; for example, hot water bags, mud packs, electric pad, and so forth. *Convective* heat is heat thrown onto the surface from some outside source, such as superheated air from electrically heated coils, etc. *Conversive* heat is energy converted into heat in the tissues themselves, by virtue of the resistance of the tissues to the passage of oscillatory high frequency or heat waves through them. Convective heat we have been using for some time in the nature of a leucodescent lamp. This heat is produced by a 500 watt electric bulb encased in a metal globe, the outlet being rather small, so as to concentrate the heat in one place. Most of the manufacturers of these various types of lamps insist, that it is not only the convective heat which is beneficial, but in addition, we obtain actinic or ultraviolet rays which play a great role in the therapeutic results. We do not look upon the small amount if any, of actinic rays as playing any role in the use of this form of heat. We have had exceedingly grateful results with this lamp in the treatment of acute sinus infections, acute otitis media, acute cellulitis, gland infections, and also in furunculosis. Given a twenty minute treatment with the lamp, we know of nothing so sedative or soothing in these acute sinus infections. We have seen beginning acute otitis media, where there is marked redness of the drum, pain and etc., disappear with one or two treatments. The infection of furunculosis disappears much more quickly, and the pain occurring is less severe, where this form of heat is used. We can recommend it very highly for a palliative treatment in these acute infections. We do not, however, wish to infer that all cases of sinus infections or otitis media can be cured by this treatment—far from it—but it has its use as above described.

The third form of heat employed is the conversive heat, and this is known as diathermia. This is divided into two subdivisions; (1) Diathermia, (2) Electrocoagulation or surgical diathermia. Diathermia consists of two forms (A) sedative, (B) stimulative. If a high degree of heat is desired, an intense hyperemia, an absorption of effusion, relaxation of muscular spasm, to sedate the pain of a neuritis, or sterilize some chronic suppurative process, then the sedative technic is employed. If it is desired to stimulate repair, irritate and stir up an intense inflammatory reaction, then the stimulation technic should be used. The details of the technic is not within the province of this paper, and can easily be obtained from any text book on electrotherapy. Suffice to state, that we have employed it quite frequently for neuralgias and pains from various other causes. Here, too, the effects are very grateful, and its use is recommended for palliative treatment of neuralgias, and to stimulate absorption of chronic inflammatory products. In surgical diathermia we employ much stronger current. The important method is a bipolar application of low tension, high frequency currents of sufficient milliamperage to coagulate and devitalize the tissue in the neighborhood of the active electrode. We employ a large indifferent electrode and a smaller active one. The effect is coagulative necrosis. The active small one is employed in two ways, viz.; (1) a small flat electrode is applied to the tissues to be destroyed, usually superficial nonmalignant growths of the skin, and (2) a sharp electrode or needle, which can be plunged into the tissue, and a coagulation necrosis occurs in the surrounding tissues as deeply as the needle is inserted. We have used this method in destruction of stumps of tonsils and in one case—a hemophiliac—a complete destruction of the tonsil. This method is slow and painful and we have discontinued its use, finding that surgery gives far better results. In malignancies, it is of very little value except to deodorize and disinfect the area, and we have also discontinued its use in these cases.

Along this same line of destruction of tissues is fulguration. This is produced by large and powerful sparks from any high frequency machine. The indifferent electrode is placed anywhere and the electrode from which the sparks are emitted is applied close to the parts at which destruction of tissues is desired. This destruction of tissues is merely superficial and

has no effect in deep growths. The only application we have made of fulguration is in the larynx after removal of benign growths, especially in multiple papilloma. In our experience, the recurrence is about the same, whether we apply to the base of these papillomata fulguration, electrocautery, alcohol or any chemical cautery, or whether we apply nothing at all. Hence, we have thrown it into discard.

Endothermia or Radiofrequency is nothing but a high frequency machine, the oscillations of which run into the millions. It is produced by the same instrument which transmits radio messages, hence the name. It is used somewhat like surgical diathermia, except that the small electrode does not coagulate the tissues but cuts through them. An indifferent large electrode is placed anywhere on the body, and the smaller one, the knife, is used as an ordinary surgical knife. The knife, when in use, is cold in the ordinary sense of the word, but it sears sufficiently to close all but the largest blood vessels. Our experience is limited to two cases, one a carcinoma of the tonsil, and the other, a carcinoma of the tongue. In the former case, the mass was resected without any bleeding, very little after sloughing, and a very quick healing. In the second case, we did a resection of half of the tongue with excellent results. These two cases are too few to enable us to speak authoritatively of its use, but nevertheless, this method is worthy of a trial.

The Percy Electrocautery consists of a number of sizes of tips, which can be heated electrically to various degrees of heat, i.e., from the glowing red to the white heat. The action produced by these tips is not electrical, but only the result of intense heat applied to the tissues, which causes a coagulation. We formerly used this method quite extensively in the destruction of malignant growths. The reaction is intense. It has no selective action upon new tissue growths, and, when used, destroys the normal as well as abnormal cells. The author of this method, Dr. Percy, advised the use of cold water jackets to control the heat and keep within the limits of its application. This we have always used, but found that its action did not control the heat. Our results with this method, as far as a cure is concerned, are an absolute failure, and we have also dispensed with this method except as a palliative agent. The electrocautery, so well known to all rhinologists and used by them for cauterizing hypertrophied

inferior turbinates, ulcers on the septum, or bleeding points anywhere, is on the same principle; viz., by use of electricity, the tip is heated to any degree of heat, and the results are due to the coagulative necrosis produced by the heat employed and not to the electric current. The use of this instrument is of great benefit if used properly and in selected cases we employ it with gratifying results. The use of the galvanocautery has for years been abandoned for cauterization in the upper straits of the nose, because of the complications arising from its use. However, realizing the heresy of such an act, one of our young associates (F.L.L.) has employed it extensively in fifty strictly polypoid cases, where radical removal of the middle turbinates and a thorough exenteration and reexenteration of the ethmoid cells have failed to check the recurrence of the polyps. These cases are purely those of the nonsuppurative, hyperplastic variety. Those cases of meningitis reported in the literature following cautery of the middle turbinate (thrombosis of the ethmoidal veins) were operated on under different circumstance than ours; namely, in the presence of a chronic suppuration. Therefore, after careful consideration, our observations indicated that many sequelae and complications which have been ascribed to this form of procedure are due to entirely different causes, and at the most, this procedure may have had only a determining influence. We feel that, by the improved technic and the careful selection of cases, this method may come into use again. However, being fearful lest its indiscriminate use lead to mishaps which would again discredit this operaton, we are working up to a larger series of cases, and will subsequently present it in detailed form.

The Electric Pulsator: There are various types of these instruments, and as we have only used one kind, we must give the results of our observation with this particular machine. All are based on the same principle, viz., an electric contrivance which creates a sort of buzzing sound. This is conducted to the ears by two rubber tubes like a biaural stethoscope, the ends placed in each external auditory meatus. At the same time, there is a politzer bag connection which the patient compresses, and this tends to mechanically vibrate the drum membrane. The electric buzz is supposed to stimulate the terminal fibers of the VIII nerve, and thereby assist the hard of hearing individual to improve his hearing. In our

experience, and we have tried it in many cases, not a single patient was benefited by this treatment in itself. As a psychic influence in some cases, it may have some beneficial result.

VIBRATORS: There are many varieties of these instruments in use today, most of which are operated by electric power. They are often used as an adjunct to some form of electric treatment, but in themselves have given us very little therapeutic results.

OZONE is a strong, irritating gas, made by passing a current of air over a high voltage generator. It cannot be used in otolaryngologic practice without combining it with some volatile oil. We employed it for several years, and while we obtained some good results, it gradually passed into disuse.

ELECTROLYSIS: Having had such poor results in the treatment of deafness resulting from chronic adhesions in the middle ear, we decided to try nascent iodin, in the hope that it might soften and absorb some of these adhesions. Knowing that the negative pole of a galvanic current has an affinity for iodin, we placed a saturated solution of iodid of potassium in the external ear, inserted an insulated copper wire electrode (positive) through the eustachian tube, and turned on a mild current. This broke up the K. I., and by the starch method, we determined the presence of iodin on the copper electrode. After trying this in a number of cases without any results, we abandoned this method of electrolysis.

We now come to another important phase of electrotherapy, viz., the LIGHT THERAPY. This form of therapy is the first ever used by mankind, for since the beginning of time, the sun with its beneficial rays has been with us. It was formerly thought that all rays were of equal length and strength, but Dr. Hertz, in 1877, discovered long invisible waves. These are now being used for the transmission of radio messages. If we split a ray of sunlight into its component parts, we find that they are divided according to their lengths. The infrared, which are not visible, are at one end of the spectrum, and are the longest; then comes the orange, yellow, green, blue, indigo, violet, which are visible, and finally the shortest of all, the invisible ultraviolet or actinic rays. In other words, the infrared, the longest waves, are the rays which produce heat. The others are the visible spectra; and the ultraviolet, the shortest and also invisible, is the one which is of greatest therapeutic value. Finsen, a Norwegian, in 1893, was the first

to make therapeutic use of the ultraviolet or actinic rays. His method was, however, very crude as compared with any of the modern quartz lamps. He obtained his actinic rays direct from the sun, but the shortest and most powerful ones were absorbed and did not reach the patient. There are, at present, two methods employed in using these actinic rays generated by a mercury lamp; one, in which a large portion of the body is treated at once, and this is done by the air cooled lamp, and the other, where more local application of the rays is desired. This is done by the water cooled apparatus, with various quartz tips. It has been shown that quartz will absorb less rays than almost any material which we can use. In using these quartz applicators, we must be sure that they are not bent more than forty-five degrees, since the majority of the rays are lost, as they travel in a straight line, but will follow the quartz applicators if not over a forty-five degree angle. In the beginning, we were told that the rays going through the applicators would be given off along the quartz, but it was soon discovered that they emanated only at the tip. The actinic rays have a penetration of only one or two millimeters below the skin or mucous membrane, as they will not penetrate through the blood. The application is followed by two reactions, the local and the general. The local is manifested by varying degrees of erythema, and the general by various blood chemistry reaction, the sum total of which may be said to be decidedly beneficial in many morbid states. It has been demonstrated that the effect of the short waves is very bactericidal. The actinic rays are being used by electrotherapeutists in many pathologic conditions, and most of them are reporting gratifying results. Our attention was called to its use in otolaryngology by a paper published in the Journal of the American Medical Association by Drs. Novak and Hollender. They reported upon a number of pathologic conditions, principally hyperesthetic rhinitis, hay fever and asthma. Their investigation shows that there is a lack of blood calcium in these individuals, which can be brought back to normal by the ingestion of calcium lactat and small doses of thyroid extract. As soon as the calcium intake was discontinued, the patients began having their former symptoms. In order to stabilize the blood calcium, they were treated by actinic rays over a varying period of time, and the results were then made permanent. We installed a quartz mercury lamp and

have been using it since. One of us (P.) treated about twenty-five cases of true hyperesthetic rhinitis by the above method. They were all given thirty general actinic treatments—most of these cases were from one to five years duration. In all of these patients, the clinical symptoms disappeared completely, and only in one did a recurrence appear. This came on suddenly about six weeks after treatment was discontinued, just as severe as ever. The patient was again placed on the treatment, and after twelve exposures, all clinical symptoms cleared up and have remained so to present time. It is now seven months since the first patient completed the treatment, the last one only a month ago. True, sufficient time has not elapsed to say that a permanent cure has been affected, but in none of them was there ever remission for so long a time. The treatment has been very efficacious in treating furunculosis, either of the ear, face or neck, as usually one or two exposures to the water cooled apparatus will abort them completely. If they are fairly well established, it will hasten the recovery. I have said nothing concerning the pathology of hyperesthetic rhinitis before and after treatment, but have only given the clinical results. We have used the rays in suppurative sinus conditions, preoperative as well as postoperative, and have had all negative results. In a few cases of asthma and hay fever, the local as well as general actinic treatments have been a failure.

Infrared Rays: These are the long invisible rays which are being used for the purpose of disinfection in deep tissues. Their penetration is considerable in contrast to the ultraviolet, or short rays. In other fields of medicine, good results are being reported, but our experience has been too limited to make any definite statement as to its efficacy.

X-ray: Regarding the use of these rays in diagnosis and treatment, volumes have been written, and it would be useless to burden you at this time with the pro's and con's of this subject. One has but to scan the literature to find glowing results from one authority and condemnation by others. As to its value in diagnosis, we must state that it is probably the most useful laboratory adjunct in the history of medicine. However, we must not lose sight of the fact that it is adjunct only, and often is at variance with our clinical manifestations; and when this does occur, we must lean toward our clinical diagnosis and not depend entirely upon our X-ray interpreta-

tions. Even in our own field, its use is so valuable and extensive, that we could not begin the discussion of its manifold usefulness without writing volumes. The rapid advancement of otolaryngology as a special field in medicine has, no doubt, in a great measure been due to the use of X-ray in diagnosis. As to its factor as a therapeutic measure, less enthusiastic reports have resulted than we formerly had anticipated. While we still employ it in some pathologic condition, in which good results are obtained, cures from its use are few and far between. In former years our great hope of its curative power was in malignancy. But we have been greatly disappointed, even with the use of the shorter rays, or deep therapy, emitted from powerful machines. We know that its action upon lymphoid tissue is selective and destructive. Its action upon embryonal cells is also destructive in nature, but when used in sufficient dosage, it also destroys healthy tissue and ultimately destroys the patient. In our personal experience, the result in carcinomas is of some value. Often it changes an inoperable case into an operable one, but rarely so. We employ it in preoperative and postoperative cases; in the former to prevent a spreading of the cancer cells, which often occurs in operative procedures; in the latter, to destroy any few cells which have migrated beyond the operative field. In our experience, we have never seen a single case of true carcinoma cured, i.e., to say when there was not a recurrence or a metastasis. In sarcomatas, we have seen good results in a small percentage of cases. Some few patients have completely recovered, and thereafter have been free from recurrences. In papilloma and surface epitheliomas, we have had many excellent results, but it is not a specific in these conditions. In treating hypertrophied and diseased tonsils, while the size may be decreased by destruction of the lymphoid portion, no relief has ever been obtained from the general symptoms due to a focal infection. We have also made use of the X-ray in treating deafness, but with absolutely negative results.

In conclusion, we believe that electricity and rays of the types enumerated above have a distinct value in our therapeutic armamentarium but their action must be thoroughly understood. The cases must be selected, and it must be remembered that one type or current, or one machine, is not a panacea for all ailments.

DISCUSSION.

DR. HAROLD M. HAYS, New York City: I think everybody who ever visited the Beck and Pollock clinic in Chicago, would realize that they are doing everything they can to try out the new methods. It is my own opinion, that all of these electrotherapeutic things are simply adjuncts that help out the treatment, whether psychologically or physically. There is no doubt that the electrotherapeutist is very enthusiastic, and yet I think he should be very conservative.

There are two classes of cases treated in which we can expect results. Those are the patients who demand treatment of some kind, and where we get our influence on the diseased condition by psychologic means. Then, there are those cases which really respond physically, where we have evident disease, such as malignant disease or some form of neoplasm.

When we come down to the real electrotherapeutic agent, we must understand that they do two things—either stimulate or destroy tissue. When it comes to stimulation, I am from Missouri. I have had all the various kinds of vibrators that have been advertised, and I have never seen a satisfactory treatment by these vibrators. Whether we will be able to get some sort of instrument in the future that will do some good, remains to be seen. In the first place, the simpler the electric instrument you use, the better results you get, and I believe the therapeutic lamp is the best. It is simple to use and has a stimulating effect. In using the quartz lamp, you will get a better effect if you use it in conjunction with analin dyes. For example, the quartz lamp therapy will not reach a cavity like the antrum by itself, but if you fill the cavity with some dye, and then insert the electrode, the ray will follow into the antrum.

There is one form of electrotherapy that Dr. Pollock did not mention, and that is zinc ionization. I think it is of great value and very simple. It is necessary to use only an ordinary rheostat connected to two electrodes, with one in the zinc solution in the cavity and the other in the patient's hand.

In the use of the quartz lamp for hyperesthetic rhinitis, I should be glad if I could show some gratifying results. I have used the quartz lamp in a series of inflammations, but I have never seen any good results from its use in hyperesthetic rhinitis, or any other acute inflammation in the nose.

I think we will go on using these electrotherapeutic measures, and whether we get our results by psychologic or physical means, it makes very little difference, provided the patient is satisfied.

DR. BERT HEMPSTEAD, Rochester, Minnesota: I have not had any experience in most of these cases, as diathermy and the different electrotherapeutic treatments are given in another department. We did, however, have a series of cases in which we tried the X-ray on the tonsils. These cases had severe diabetes, and were cases in which we did not operate because we were afraid of coma. This was before the days of insulin. These cases were exposed to the X-ray, but no benefit obtained. After insulin came out, the tonsils were enucleated and they showed no change, either bacteriologically or histologically. In other departments

of the clinic, the different lamps are used, but in our department we have had no experience.

Dr. Samuel Salinger, Chicago: The apparent discrepancy between the results of the quartz lamp in hyperesthetic rhinitis in the essayists' series as compared with those obtained by observers in other parts of the country, seems to me to be due to the fact that in Chicago hayfever has been milder this summer than for ten years past. Several patients whom I had formerly treated reported no symptoms at all this year.

In crediting a method of treatment with cures, therefore, one must not lose sight of the unusually favorable climatic conditions.

Dr. Joseph C. Beck, Chicago: That last remark was very unkind, because seven months ago we did not have any hayfever, and these cases extended farther back than that. I will say this, however, Dr. Pollock said one of us had experience with twenty-five cases. I have observed more cases than that treated by that method, but they were not brilliant results. He observed these cases himself, and the results are different from mine.

Let me say this in regard to electrotherapeutic measures, that we must use different methods and try them out and accept or condemn them. The only objection I have to all this apparatus, is that it is at the command of the socalled quack, who uses it in his office. Patients say, "Dr. So-and-So has one of those machines," and we are likely to be thrown into that category.

TONSILLECTOMY AND ADENOIDECTOMY IN SELECTED CASES DURING THE PERIODS OF PRIMARY AND SECONDARY INFECTIONS, A RATIONAL AND CONSERVATIVE PROCEDURE. REPORT OF CASES.

FREDERICK T. CLARK, M.D., F.A.C.S.

WESTFIELD, MASS.

At the outset, in order to forestall any misapprehension, and although indicated in the title of this paper, the writer wishes to state he does not advocate indiscriminate tonsilloadenoidectomy. He believes a careful examination into the condition of each patient should precede the selection of all cases submitted to operation.

This discussion includes all tonsillectomies and tonsilloadenoidectomies done under local or general anesthesia:

(1) When tonsils or adenoids are acutely inflamed or infected, as during any kind of an angina; or

(2) When the operation is done in the course of one of the acute contagious diseases; or

(3) When the operation is done during the course of some secondary infection, as during an acute otitis or mastoiditis, bronchitis, nephritis, endocardits, neuritis, rheumatism, or any other secondary infection that may be due to infected tonsils or adenoids as the primary focus of infection.

No claim is made for the originality of the matter here presented. It is believed however, that the discussion will serve to focus attention on the importance of tonsilloadenoidectomy, in the conditions mentioned, and states the belief that what is now looked on askance by many practitioners as a rash and radical procedure, will, at no distant day, be regarded as rational and conservative, yes, even life saving, surgery.

During the past few years, there have been numerous references in the literature to the removal of tonsils, and of tonsils and adenoids, during the period of acute infections. Until recently, to have advocated tonsillectomy and adenoidec-

tomy during the period of acute infections for the relief of any but emergency conditions would have been received as a therapeutic heresy. At the 1923 session of this Academy in Washington, we were told that this organization is the largest and most representative body in the world, of specialists in any department of medicine and surgery. At that session, two papers were read and very generally discussed, in which the essayists, Thomas[1] and Wolfe[2], urged, when indicated, the removal of the tonsils and adenoids when acutely infected. Porter[3] has recently reported a number of cases operated during the periods of acute infection. He concludes there are two general types of peritonsillar abscess; the common supratonsillar type, and the basilar. He states that any treatment or operative procedure other than enucleation in the latter type is dangerous to the life of the patient. He believes, too, that marked glandular swelling following acute tonsillitis in children is a strong indication for prompt enucleation, and Porter predicts, without any qualifications, that it will ultimately be considered proper surgical procedure to remove tonsils during acute attacks.

Thomas and Wolfe, in Washington, urged, when indicated, the removal of tonsils and adenoids when acutely infected. Tonsilloadenoidectomy was urged by Thomas, in selected cases, as one of a series of steps in the management of acute otitis media, and prevention of the acute mastoid operation. Thomas states he has many times removed tonsils and adenoids in adults when they were in a state of severe acute inflammation, and has had no cause to regret the operation. Such patients recover much more promptly following operation than when left for the acute inflammatory process to subside before operating, when a period of from one to three weeks or even longer may be required, during which time the patient is absorbing toxins.

Wolfe, in his paper before the Academy on the prevention of surgical mastoiditis, comes out flatly and says the prompt removal of tonsils and adenoids, together with free incision of the ear drum, is his first consideration in acute catarrhal otitis media (earache), and will often prevent acute purulent otitis and mastoiditis from developing. Dean[4] and others have called attention to the almost constant presence of infected tonsils and adenoids in children with sinusitis. Wolfe says that probably ninety-five to ninety-eight per cent of all

cases of acute purulent otitis media occurring in children can be directly or indirectly attributed to infected tonsils and adenoids.

I wonder if we have a sufficient realization of the importance of Chadwick's[5] recent contribution to medical knowledge? Has it penetrated our consciousness that his conception of juvenile tuberculosis begins a new epoch in the handling of the white plague, and will save multitudes of lives that would otherwise be offered up on the altar of ignorance? Do we appreciate the fact that the Westfield Sanatorium has become a Mecca to which internists, pediatricians and roetgenologists, alike interested in this new conception of juvenile tuberculosis, are coming for instruction? Chadwick's conception of the juvenile type of the disease points to the tonsils and adenoids as among the portals of entry for the tubercle bacilli, which eventually invade the bronchial glands. In cases of this type, our first consideration should be to determine, by means of the tuberculin test, whether we have to deal with a tubercular or other form of infection. In either case, competent advice should be sought as to an immediate tonsilloadenoidectomy.

That infected tonsils and adenoids in children are responsible for a large percentage of the cases of bronchitis and bronchopneumonias, has become to pediatricians and otolaryngologists a commonplace. This fact, however, does not seem to be well recognized by the family physician. In the writer's experience many children, even of physicians themselves, who are subject to bronchial irritation, coughs and colds, are treated without recognition of, or recommendation for the removal of the primary infection in the throats of these little patients.

So far as I have learned, Glogau[6], of New York City, in 1920, was among the first well known otolaryngologists to urge the immediate radical removal of infected tonsils and adenoids as of prime importance in impending, or present, acute purulent otitis and beginning mastoiditis. The bitter criticisms of Glogau's contentions are matters of record. After an observation extending over many years, during which the writer practiced these procedures in many cases where it was possible to gain the consent of the parents and family physician, he has no hesitancy in aligning himself with the men who have taken this advanced position. With Glogau, he

believes the mastoid operation before long will become a comparatively rare occurrence, because, due to appropriate procedures and prophylaxis, it will be necessary only in exceptional cases.

The writer goes still farther, and believes the time is not far distant, when the tremendous conservatism which has heretofore looked askance at any operative procedures in acute contagious diseases will give way to a more rational and favorable attitude toward tonsillectomy and adenoidectomy in these conditions.

In 1922, Place[7], of Boston, pointed out the importance of the tonsils (and adenoids) as points of attack and portals of entry in scarlet fever and diphtheria. It is well known that throat symptoms in scarlet fever occurring in tonsillectomized patients are almost invariably slight, even to the point of absence. Severe faucial diphtheria is excessively rare in tonsillectomized patients, and when it does occur in such cases, it is posterior to the posterior pillars. The writer wishes to point out the rarity, in his experience, of measles, in all but its mildest types, in patients who have had their tonsils and adenoids removed.

The studies of Dochez[8] concerning the significance of the streptococcus hemolyticus in scarlet fever, indicate that this organism found in the throats of scarlet fever patients may be a specific type, readily distinguishable by agglutination and other immunologic tests from the types of streptococcus hemolyticus causing other forms of angina and general septic conditions. This apparent specific type of organism was present in all of a large series of cases of scarlatina. Dick and Dick[9], of the John McCormick Institution for Infectious Diseases in Chicago, after many years of research work in this field, have strengthened the evidence in favor of a specific strain of the hemolytic streptococcus as the cause of scarlatina. They have made extensive studies looking toward the development of an antitoxic serum. Their work has been confirmed by Zingher[10] and by Branch and Edwards[11], working independently, and it is confidently expected that the time is near at hand, when the susceptibility of individuals to scarlet fever will be generally recognized by a procedure similar to the Schick test in diphtheria, and when susceptible, they will be rendered immune as is done today by toxin antitoxin in diphtheria.

In view of the inference, then, that the tonsils and adenoids are the chief foci of infection in scarlet fever, diphtheria and measles, it is only logical that tonsilloadenoidectomy be practiced, not only as a prophylactic measure, but as a rational means of shortening contagion during acute attacks, in selected cases, and to prevent the frequent otitic and nephritic sequelae. Taken in conjunction with the administration of Dochez's antiscarlatina serum, as reported by Blake[12], the outlook for the prevention of scarlet fever, and its cure when developed, seems bright indeed. There is no doubt that a much more favorable attitude toward tonsilloadenoidectomy in the acute infectious diseases has been developed in the past few years. Not only as a prophylactic measure is it becoming a recognized procedure, but operation is now being urged by many pediatrists during the active stages of the acute contagious diseases. Place reports one hundred and twenty-two cases of scarlet fever operated during the active stages of the disease. Many of these cases were operated because of the continuation of the contagiousness of the patients, and because complications were threatening before operative intervention was considered. Of this series, he reports seventy-three and seven-tenths per cent recovering with the development of no complication. When we think of the frequency of albuminuria and purulent otitis as sequelae of scarlatina, the significance of this report is worthy of our earnest consideration. His operated cases varied in age from six months to twenty-five years. During diphtheria, in diphtheria carriers, septic sore throat, tonsillitis (chiefly to cut short an acute arthritis or rheumatoid attack) and in chorea, tonsillectomy was performed seventy-four times. Place comes out strongly, and says tonsilloadenoidectomy is a valuable means of shortening the contagiousness of scarlet fever and diphtheria in suitable cases; that there is reason to believe early operation in scarlet fever tends to reduce the danger of complications; the operative dangers are not great and local infection is less common than in nonoperative cases.

The writer is of the impression that the average practitioner has a clear conception of infected tonsils and adenoids as among the chief portals of entry and foci of rheumatic infections. Starling[13] points out that the presence of enlarged lymphatic glands under the jaw is more constant proof of tonsillar infection than the appearance of the tonsils. Ton-

sillar infection not only initiates the onset of rheumatic conditions, but may directly prolong an acute attack and be the cause of repeated subsequent attacks. He urges the removal of the tonsils, at once, to end an acute attack and to prevent further infection, and he stresses the importance of removing every particle of tonsillar tissue. A mere tag or stump left behind may be as potent a source of infection as were the whole tonsils before operation. In the course of rheumatic infection, the earlier the tonsils are removed, the greater the benefit derived. The benefits are manifested not only in the acquisition of a healthy color and an increase in weight and efficiency, but also in the prevention of further infection of the endocardium and an apparent mitigation of the damage already suffered by it. The operation also removes an infective focus, the toxin from which is prejudicial to the heart muscle as well as to the whole system.

Only a little more than a year ago, Cautley[14] indicated, that it is not widely recognized that infection of the tonsils in little children and adults is frequently the explanation of an attack of hematuria, albuminuria, and acute nephritis, which becomes chronic, and that tonsilloadenoidectomy is a necessary procedure, that to be life saving must be done before structural changes in the kidneys have developed. At present, it is a moot question whether the enucleation should be done at once or wait until the local infection or hematuria has subsided. In severe cases, or those tending toward chronicity, we should have no hesitation in urging immediate operation.

Only a little more than a year ago, Smith and Bailey[15], in the Archives of Pediatrics, reported several cases to demonstrate the close relationship between infected tonsils and infected kidneys, and also to show the result obtained by removal of the tonsils in the acute stages of the nephritis or pyelonephritis. These authors say: "As the tonsils (and adenoids) are perhaps the most common avenue of infection, it is logical, in a seemingly primary acute nephritis or pyelonephritis, first to suspect that the tonsils are responsible for the entrance of the infecting organisms. If the tonsils are found chronically infected (culture), and no other focus is discoverable, the tonsils (and adenoids) should be immediately removed, even in the acute stage of the kidney disorder, to prevent permanent structural changes in the kidneys."

It is well recognized by ophthalmologists that obscure inflammations of the eye are most frequently the result of a focal infection. Bell[16], of New York, has brought prominently to our minds the relations of teeth, tonsils and intestinal toxemias to diseases of the eye. He speaks of "The ramifications of the three T's" (teeth, tonsils and toxemias of the intestinal tract) as being so closely identified in his mind, as etiologic factors, that in thinking of any one of the three T's in connection with obscure eye diseases, the other two are invariably brought to mind.

Before summing up the conclusions that are to be logically formulated from this discussion, the following brief report of typical cases is included. The writer regrets he is unable to report cases that are representative of each type of primary and secondary infection previously referred to. He believes, however, that this inability does not materially detract from the logic of the opinions herein expressed.

Case 2183. Mr. S. G. A., 38, referred May 8, 1924 by Dr. J. B. Atwater. Patient in bed. For weeks had been having intermittent attacks of sore throat, mostly on the right side, and at intervals coughed and raised bloody sputum. Five days previously, developed severe sore throat on right side. Had taken but little food, and painful swallowing was apparent. Voice muffled. Glands under right side of jaw much swollen. Right tonsil patched, swollen, soft and spongy. Right tonsillar region very tender. Definite fluctuation could not be made out. Left side of throat was not involved. Patient was coughing and raising much pus, that could be readily seen on posterior pharyngeal wall. This patient had served actively four years under great strain in the British aviation service. His plane crashed, and after nine months of hospitalization, he returned to civil life with nerves badly shattered. His tonsils had been operated prior to his entry into the army. He dressed, was driven to my office, where, under local anesthesia, the right tonsil was enucleated. It was a large, flat, spongy, submerged tonsil, and its enucleation exposed an abscess cavity between the base of the tonsil and the musculature forming the tonsillar fossa. He was returned home and to bed. He made an uninterrupted recovery, and in two weeks appeared at my office for removal of the left tonsil. This patient, during convalescence from the first tonsillectomy, was referred to Dr. Chadwick for examination of his

lungs. Pulmonary abscess had been suspected by Dr. Atwater, and I believe the physical signs and X-ray plates, while not positive, pointed to that diagnosis as the cause of his cough and expectoration. At any rate, he has made a complete recovery and reports he has not felt as well since his entry into military service.

Case 2235. Ralph S. W., 24, referred by Dr. G. H. Janes, June 24, 1924. Four days before, woke up with stiff neck and sore throat, that as the day wore on became more painful. Two days later, saw his local physician, who painted his throat. The next morning, his throat was no better. The glands under the angle of his right jaw were swollen and tender, and the area below the ear and over the sternocleidomastoid muscle swollen. Right ear felt stuffy. The right tonsillar region was swollen, the pillars of the right side edematous and tonsillar follicles grayish. The tonsil was very tender on pressure, and dysphagia was a prominent symptom. There was no swelling of the left side, but the tonsil was patched and submerged. Temperature 99.4° F. Pulse 84. Under local anesthesia did immediate tonsillectomy, exposing a small abscess containing a few drops of thick, foul smelling pus. The abscess was between the base of the tonsil and the musculature of the tonsillar fossa. The temperature and pulse the next morning were normal. The cervical swelling had disappeared, and the glandular tenderness was much less noticeable. He made an uninterrupted recovery, and one week later drove his car to his home, a distance of twenty miles.

Case 715. Helen A. H., 4, referred by Dr. A. J. Logie, May 28, 1922. In March, this little girl was ill with influenza. Her father stated she had been miserable ever since. About ten days prior to my seeing her, she developed earache with discharge from both ears. Her father was urged by her local physician to take her to the hospital in Pittsfield for double mastoid operation. She was then seen by Dr. Logie, who referred her to me. On examination, right mastoid tender on pressure, but not swollen, red or edematous. There was pus in the canal, which was swollen, and a pouting perforation in the roof of the canal was seen near the drum. The left mastoid was tender but not swollen, red or edematous. There was pus in the canal, and a perforation in the drum could be seen. Temperature was 103° F. and pulse 130. She was

emaciated and septic. She was sent to the hospital, and immediately under ether by Dr. Schoonmaker, both drums were freely incised and tonsilloadenoidectomy done. In four days time all tenderness of mastoids had disappeared. She made a slow but uninterrupted recovery. Due to her sepsis, it was three weeks from the date of her operation before she kept a normal evening temperature. On the twenty-sixth day, both ears were dry, and the next day she went home.

Case 2219. Jessie E. S., 13, referred by Dr. M. D. Chisholm, June 16, 1924. Gives history of having frequent attacks of tonsillitis. Glands under jaw palpable. Four days ago, without apparent cold in head, developed left earache which continued, with the development of pain in the head behind the ear. On examination, the left canal is swollen and the drum red. The mastoid is very tender on pressure over the antrum. Tonsils hypertrophied. Temperature 99.4° F. Pulse 100. She was sent to the hospital and immediately, under ether anesthesia by Dr. Chisholm, free incision of the drum was made and a thorough tonsilloadenoidectomy done. A profuse serosanguinous fluid welled out of the ear on incision of the drum. The next morning the mastoid tenderness had disappeared, and she returned home the following day. She made a speedy uninterrupted recovery.

Case 2256. C. M., male, 14 months, referred July 16, 1924, by Dr. W. L. Connery. Mother states the child has been crying at intervals for the past ten days with earache. Says he has had a cold (snuffles) ever since he was born. He grew rapidly 'till eight months old, and has not gained an ounce in the past six months. Is feverish and fretful at night, and sleeps much of the day. Temperature at three P. M., when examined, 101.6° F. Glands under angle of jaw swollen and tender. Both ear drums dusky and lack luster. Tonsils patched, and mass of adenoids palpated. Under ether by Dr. Connery, immediate tonsilloadenoidectomy was done. Went home the next morning, and forty-eight hours later, the child's temperature was normal and he slept all night. He has made a complete recovery and is rapidly gaining weight.

Case 1676. Marion C. T., 4, referred by Dr. J. B. Atwater, June 15, 1918, for removal of tonsils and adenoids. This advice was not immediately followed. Four days later, she developed left ethmoiditis, with swelling and edema of the left eye and proptosis. Right side not affected. The nose

lungs. Puimonary abscess had been suspected by Dr. Atwater, and I believe the physical signs and X-ray plates, while not positive, pointed to that diagnosis as the cause of his cough and expectoration. At any rate, he has made a complete recovery and reports he has not felt as well since his entry into military service.

Case 2235. Ralph S. W., 24, referred by Dr. G. H. Janes, June 24, 1924. Four days before, woke up with stiff neck and sore throat, that as the day wore on became more painful. Two days later, saw his local physician, who painted his throat. The next morning, his throat was no better. The glands under the angle of his right jaw were swollen and tender, and the area below the ear and over the sternocleidomastoid muscle swollen. Right ear felt stuffy. The right tonsillar region was swollen, the pillars of the right side edematous and tonsillar follicles grayish. The tonsil was very tender on pressure, and dysphagia was a prominent symptom. There was no swelling of the left side, but the tonsil was patched and submerged. Temperature 99.4° F. Pulse 84. Under local anesthesia did immediate tonsillectomy, exposing a small abscess containing a few drops of thick, foul smelling pus. The abscess was between the base of the tonsil and the musculature of the tonsillar fossa. The temperature and pulse the next morning were normal. The cervical swelling had disappeared, and the glandular tenderness was much less noticeable. He made an uninterrupted recovery, and one week later drove his car to his home, a distance of twenty miles.

Case 715. Helen A. H., 4, referred by Dr. A. J. Logie, May 28, 1922. In March, this little girl was ill with influenza. Her father stated she had been miserable ever since. About ten days prior to my seeing her, she developed earache with discharge from both ears. Her father was urged by her local physician to take her to the hospital in Pittsfield for double mastoid operation. She was then seen by Dr. Logie, who referred her to me. On examination, right mastoid tender on pressure, but not swollen, red or edematous. There was pus in the canal, which was swollen, and a pouting perforation in the roof of the canal was seen near the drum. The left mastoid was tender but not swollen, red or edematous. There was pus in the canal, and a perforation in the drum could be seen. Temperature was 103° F. and pulse 130. She was

emaciated and septic. She was sent to the hospital, and immediately under ether by Dr. Schoonmaker, both drums were freely incised and tonsilloadenoidectomy done. In four days time all tenderness of mastoids had disappeared. She made a slow but uninterrupted recovery. Due to her sepsis, it was three weeks from the date of her operation before she kept a normal evening temperature. On the twenty-sixth day, both ears were dry, and the next day she went home.

Case 2219. Jessie E. S., 13, referred by Dr. M. D. Chisholm, June 16, 1924. Gives history of having frequent attacks of tonsillitis. Glands under jaw palpable. Four days ago, without apparent cold in head, developed left earache which continued, with the development of pain in the head behind the ear. On examination, the left canal is swollen and the drum red. The mastoid is very tender on pressure over the antrum. Tonsils hypertrophied. Temperature 99.4° F. Pulse 100. She was sent to the hospital and immediately, under ether anesthesia by Dr. Chisholm, free incision of the drum was made and a thorough tonsilloadenoidectomy done. A profuse serosanguinous fluid welled out of the ear on incision of the drum. The next morning the mastoid tenderness had disappeared, and she returned home the following day. She made a speedy uninterrupted recovery.

Case 2256. C. M., male, 14 months, referred July 16, 1924, by Dr. W. L. Connery. Mother states the child has been crying at intervals for the past ten days with earache. Says he has had a cold (snuffles) ever since he was born. He grew rapidly 'till eight months old, and has not gained an ounce in the past six months. Is feverish and fretful at night, and sleeps much of the day. Temperature at three P. M., when examined, 101.6° F. Glands under angle of jaw swollen and tender. Both ear drums dusky and lack luster. Tonsils patched, and mass of adenoids palpated. Under ether by Dr. Connery, immediate tonsilloadenoidectomy was done. Went home the next morning, and forty-eight hours later, the child's temperature was normal and he slept all night. He has made a complete recovery and is rapidly gaining weight.

Case 1676. Marion C. T., 4, referred by Dr. J. B. Atwater, June 15, 1918, for removal of tonsils and adenoids. This advice was not immediately followed. Four days later, she developed left ethmoiditis, with swelling and edema of the left eye and proptosis. Right side not affected. The nose

was full of mucopus (snuffles). Temperature 101°+, pulse 140+. The parents would not consent to operation, which would, I believe, have saved this little girl much suffering and illness. Under the use of nasal irrigations, argyrol instillations in nose and hot compresses to the eye, the swelling finally entirely subsided without the development of an orbital abscess. The treatment was kept up pretty continuously throughout the summer, until, when seen again September 16, the swelling had entirely disappeared. There was, however, a profuse purulent discharge from the nose, indicative of the involvement of the ethmoids. The next day tonsilloadenoidectomy was done. The writer at this time entered the army and did not see the child again for more than two years. She had been very much better in the two preceding years since the tonsilloadenoidectomy. However, she gave the history of frequent colds and of persistent cough during the period of these coryzas. For several days, beginning with a cold in the head, she had coughed persistently, keeping the household awake and dancing attendance. Examination of right nostril was negative. The left middle turbinate was swollen and in contact with the septum. Transillumination showed marked shadow of left antrum. Larynx was negative. The child had a left purulent ethmoiditis and maxillary sinuitis. Immediate operation was advised and declined. The writer did not see the patient again for more than three years, December 11, 1923, when she was referred by Dr. Chadwick. She had developed profuse purulent discharge from the right as well as the left nostril, and had a violent and almost continuous cough. She had been under treatment in a neighboring city with negative results, and had been finally taken to Dr. Chadwick to determine if the cough was of tubercular origin. The tuberculin test was negative, but the X-ray showed very large bronchial glands about the hilum of the lungs. Examination of nose revealed double purulent ethmoiditis and left frontal and maxillary sinuitis. Two days later at the hospital, under warm ether vapor anesthesia by Dr. E. S. Smith, double middle turbinectomy, partial exenteration of right ethmoid, and puncture and irrigation of much pus from left maxillary sinus was done. She made prompt recovery and went home the next day. Was sent to Florida for the winter. Gained seven pounds. Lost her cough. Returned home in April free from frontal or antral infection,

and has been gaining in weight and health since her return. This case has been cited in detail as the story of what quite likely might have been avoided if the original foci of infection had been promptly removed as advised six years before.

CASE 57. Mrs. H. W. A., 47, referred by Dr. G. H. Janes for examination of throat. For past three years she has had much lameness and stiffness of her muscles. Her throat had been sore, dry and irritable for several months, with stiffness of muscles of the neck. On examination, cheesy masses were extruded from the tonsils, and the left tonsil showed swollen patched follicles. Under ether by Dr. Janes, tonsillectomy was done. No adenoids present. Two years later, this patient reports much improvement of her rheumatoid symptoms, with no sore throat.

CASE 2282. Mrs. F. S. B., 65. Referred by Dr. G. H. Janes, August 21, 1924. For forty years or more, has had almost yearly attacks of tonsillitis, often followed by quinsy. Her most recent attack of peritonsillar abscess occurred last May. She has had two attacks of tonsillitis since May, and when I saw her, she was recovering from the last attack. Both tonsils were dark red, as were the overlying pharyngeal pillars, while the left tonsil was much more enlarged than the right, protruding into the pharynx. For a number of years, following her attacks of tonsillitis, her knees have been swollen, lame and tender, but the arthritic symptoms cleared up pretty well each time after a few weeks lameness. However, since her attack in May, the joints of her fingers, wrists, elbows, knees, ankles and toes have been swollen, tender and painful. Her heels have been so tender, and the lower lumbar region so lame, that she has been able to get around the house with difficulty. The two attacks of tonsillitis that have occurred since May have added to her discomfort and disability. This patient was brought to my office, and, under local anesthesia, careful thorough tonsillectomy was done. At this writing, two days after operation, she is resting comfortably and declares her throat not nearly so sore as it has often been with tonsillitis. She is using salicylates as a gargle and internally, and says her joints are much more comfortable. Of course it is too early to forecast the results in this case.

CASES 2201, 2267 and 2268, seen July 23, 1924, are reported as a group of three children of the same family, with ages ranging from four to nine years. All were convalescing from

scarlet fever, desquamation of the palms and feet not completed. One had an acute arthritis of ankles, knees and wrists and foul discharge from both ears. All had temperatures above 100° F. each evening, enlarged, tender cervical glands, which in one child were as large as English walnuts. Cultures from each throat showed the hemolytic streptococcus and micrococcus catarrhalis. Urinary examinations in each case was negative. Under warn ether vapor anesthesia by Dr. G. H. Janes, at Noble Hospital, the throats of these patients were relieved of infected tonsils and adenoids. They returned home the following day, and made an uninterrupted recovery, all being out of doors at quiet play the day following their return from the hospital. In two weeks time, all discharge from ears, arthritis and enlarged glands disappeared, and all three patients had gained much flesh. The parents were highly delighted with the progress made by these children.

Case 885. E. B. L., 34, referred by Dr. J. B. Atwater, April 2, 1923. For ten days had a severe head cold followed by inflamed eyes. The right eye cleared up, but the left one showed a sluggish iritis. The iritis did not clear up under the usual treatment. A superficial keratitis developed, and the eye became very red. The vision was reduced to 20/70 Nearly three years before, I had treated this patient for sinus trouble and had then recommended the removal of his tonsils. As his eye symptoms did not clear up under the usual treatment, tonsillectomy was done, and a periapical alveolar abscess was evacuated by extraction of a tooth. The eye symptoms, which had persisted over a period of nearly four months, cleared up within a week.

Conclusions.

1. In certain types of acute angina, with threatening or developed atypical peritonsillar abscess and marked glandular involvement, immediate tonsilloadenoidectomy should be done.

2. In acute or chronic otitis, with or without mastoid involvement, if pathologic tonsils and adenoids can be demonstrated, their prompt removal is one of the first considerations, among other measures, that should be taken to cure the otitis and abort a surgical mastoiditis.

3. Infected tonsils and adenoids should be removed as a first step in clearing up any sinuitis.

4. Infected tonsils and adenoids should be removed as a first step in children subject to coughs, colds, bronchitis and bronchopneumonia.

5. In suspected or present juvenile tuberculosis, removal of infected tonsils and adenoids should be a first consideration.

6. As a prophylactic measure against acute contagious and exanthematous diseases in children with pathologic tonsils and adenoids, their early removal is indicated; and in selected cases, for the purpose of aborting or cutting short a threatening or present secondary infection, tonsilloadenoidectomy may be used as a rational, even life saving procedure. It may also be wisely employed as a means of shortening the period of contagion.

7. In unaccounted for hematuria, albuminuria, acute nephritis or pyelonephritis, the tonsils and adenoids should be suspected as the focus of infection, and if not above suspicion, prompt tonsilloadenoidectomy should be done to prevent structural changes in the kidneys.

8. In rheumatic or arthritic infections, or neuritis and certain types of goitre, if the tonsils and adenoids are not above suspicion, tonsilloadenoidectomy is indicated for the removal of an infective focus prejudicial to the muscular, nervous and circulatory systems.

9. In obscure unaccounted for eye diseases, the tonsils and adenoids should not be overlooked.

10. Tonsilloadenoidectomy, if carefully done, in selected cases, during primary and secondary infectious attacks, is not attended by grave dangers to the patient but is a procedure of value to patient and surgeon alike.

REFERENCES.

1. Thomas. Transactions Am. Acad. Oph. and Oto-Laryngology, 1923, p. 372.
2. Wolfe. Transactions Am. Acad. Oph. and Oto-Laryngology, 1923, p. 376.
3. Porter. Boston Med. and Surgical Jour., April 24, 1924, vol. XVII, p. 693.
4. Dean. Southern Med. Journal, Oct., 1922, vol. XV, p. 846.
5. Chadwick. Boston Med. and Surgical Journal, August 7, 1924, p. 252.
6. Glogau. Laryngoscope, January, 1920, vol. XXX, p. 83.
7. Place. Boston Med. and Surgical Journal, September 21, 1922, vol. CLXXXVII, p. 434.
8. Dochez. Proc. Soc. Exper. Biol. and Med., January, 1924, vol. XXI, p. 184.
9. Dick and Dick. Jour. Am. Med. Assn., January 26, 1924, vol. LXXXII, pp. 265, 301. Jour. Am. Med. Assn., July 12, 1924, vol. LXXXIII, pp. 2, 84.

10. Zingher. Proc. Soc. Exper. Biol. and Med., March, 1924, vol. XXI, p. 293.
11. Branch and Edwards. Jour. Am. Med. Assn., April 19, 1924, vol. LXXXII, p. 1260.
12. Blake. Boston Med. and Surgical Journal, July 10, 1924, vol. CXCI, p. 43.
13. Starling. Guy's Hospital Reports, London, October, 1923, vol. LXXIII, p. 388.
14. Cautley. Arch. Pediat., February, 1923, vol. XL, p. 84.
15. Smith and Bailey. Arch. Pediat., August, 1923, vol. XL, p. 525.
16. Bell. Am. Journal of Oph., February, 1924, vol. VII, p. 118.

DISCUSSION.

DR. HARRIS P. MOSHER, Boston: The writer starts his paper by apparently waving the red flag of radicalism, but when he comes to the responsibility of drawing his conclusions, they are so conservative that I for one have no argument with him. I agree with him. From the standpoint of logic he is right. Knowing the source of infection in any given case, the thing to do is to remove the source as soon as it can be safely done. Evidence is rapidly accumulating that this can be done in very many cases.

It is not good surgery to operate in the presence of acute infection. All surgeons prefer to do an interval appendectomy. If the infection shows signs of getting beyond the appendix, then immediate operation is imperative. The analogy between operations on the tonsil and the appendix is very complete. Given a tonsil which is damaging the heart or the kidneys, an operation during the acute infection is advisable, because the damage inflicted is done quickly and is often safer. In joint conditions, I think the surgeon can in most cases safely wait.

Pus between the tonsil and its surgical capsule is likely to give general sepsis. Porter has shown that the removal of the tonsil during the acute infection is a safe procedure. Barnes has shown that removal of the already nearly detached tonsil in peritonsillar abscess is safe. Other observers have gotten into trouble from the spread of sepsis after this procedure.

The work of Place is of the greatest importance. To shorten the course of scarlet fever and to prevent its complications is a great advance. This advance will, in my opinion, stand unless Dick makes it unnecessary with his antitoxin.

I agree with the writer, that operation in selected cases will be done more and more. Experience has proved that it can be safely done. In my opinion, we have taken tonsil operations both too lightly and too seriously—too lightly, when we make an office operation of adult tonsilectomy and let the patient get out of touch with us in a few days; too seriously, when we delay operation in the face of far flung infection.

DR. ROSS H. SKILLERN, Philadelphia, Pa.: I will take up conclusion 1: "In certain types of acute angina, with threatening or developed atypical peritonsillar abscess and marked glandular involvement, immediate tonsilloadenoidectomy should be done."

Regarding the removal of tonsils in acute conditions, I do not know. I am perfectly willing to be shown, but it looks to me, on the face of it,

a questionable procedure. I can readily see, where you have a pair of enormous tonsils with swelling that interferes with respiration, outside of the fact that they are badly inflamed, why it would be necessary to remove them. Then take peritonsillar abscess, if you will. Many years ago, the late, lamented Dr. William Ballenger, of Chicago, showed us that the removal of a tonsil with a large abscess behind it was the proper thing to do, and without serious injury. I am not at all sure but what he was right, because it is half dissected out anyhow, and you have to take it out some time or other. On the other hand, suppose we have an acute infection in the throat with measles or diphtheria or scarlet fever. Here you have a number of local manifestations of systemic disease. Under these conditions, I think it would be exceedingly dangerous to do any surgery in the throat. I have in mind a case in Philadelphia, a two months old child of a very prominent citizen. Two weeks after an attack of measles, the tonsils were removed, which was quickly followed by double otitis media, meningitis, brain abscess and death. So I think that with acute exanthemata, where we have a local manifestation of systemic condition, it is probably unwise to use any surgery in the throat.

His third conclusion is: "That infected tonsils and adenoids should be removed as a first step in clearing up any sinusitis."

I merely read this to place myself emphatically beside the writer in his conclusions. Dean, in his work with children, has shown conclusively that a great many cases of sinusitis can be cured by the simple removal of tonsils and adenoids, without any other treatment whatever, so that it has now become a custom to remove tonsils in sinusitis in children.

His eighth conclusion is: "In rheumatic or arthritic infection, or neuritis and certain types of goitre, if the tonsils and adenoids are not above suspicion, tonsilloadenoidectomy is indicated for the removal of an infective focus prejudicial to the muscular, nervous and circulatory systems.

Nearly all cases of adults come under this category. In adults we seldom operate for any local condition; it is always some remote condition—heart or kidneys or joints or what not. In these cases, operation is not done in spite of the condition, but actually to remove the cause.

His ninth conclusion is: "That in obscure unaccounted for eye discases, the tonsils and adenoids should not be overlooked."

I do not know whether there is much in this or not. I have been bitterly disappointed in the eye complications clearing up after tonsillectomy: It has been my experience that 90 per cent of the eye condititons I see and cannot trace to something else come from the accessory sinuses of the nose, and it finally comes to the opening of these sinuses before we get any relief from the symptoms. I must reiterate, that I have been bitterly disappointed in the eye symptoms clearing up after surgery for the removal of tonsils.

Dr. Joseph C. Beck, Chicago: How much importance does the essayist place on the question of the anesthetic given during these acute cases, as to the danger from anesthesia? Also please tell us something about whether the blood examination plays any important role in your indications. We know that in some of these conditions there is a peculiar type of blood picture.

Dr. Frederick T. Clark, Westfield, Mass. (closing): In answer to Dr. Beck's query, in regard to cases of atypical peritonsillar abscess, these have been done under local anesthesia, using 1 per cent procain, and without any disagreeable consequences.

In regard to the blood picture, I have had little experience. In the matter of contagious diseases, we have a series of three cases of scarlet fever that I have operated, but no blood examination was done further than having the coagulation time taken. We did make a swab of the tonsils, and found of course the streptococcus hemolyticus, but no study of the blood has been made in any of these cases.

In closing, I wish to thank Doctors Mosher and Skillern for their courtesy and fairness in discussing my paper.

DEMONSTRATION OF NEW INSTRUMENTS AND PRESENTATION OF CASES.

MOTOR DRIVEN ANTRUM DRILL.

ROY S. BINKLEY, M.D.

DAYTON, OHIO.

In presenting this motor driven antrum drill to the Academy for your inspection, I have in mind an instrument that can make an opening in the antrum with the least amount of resistance and disturbance to the patient, and accomplish more than trocar and rasp in the same amount of time. This instrument is simple of construction, easy to handle, and is used under direct vision. The opening into the antrum is made more anteriorly, on the floor of the nose, and at the same time the opening can be made as large as desired without changing or withdrawing the burr. This drill cannot be lost in the antral cavity, nor is there danger of breaking through, catching or sticking; because, in the event of this happening, the multiple friction drive disc located in the handle will disengage the burr until released or the instrument is withdrawn. It can be sterilized the same as any surgical instrument.

1. HANDLE AND CANULAE FOR SUCTION AND IRRIGATION.

JOS. C. BECK, M.D., HARRY L. POLLOCK, M.D., AND FRANCIS L. LEDERER, M.D.

CHICAGO.

The application of suction in the treatment of diseases about the head and neck has long been carried out, but to employ it as a definite therapeutic measure has not been often emphasized. The suction, as employed in our clinic, has been of the capillary type, and various mechanical devices and forms of capillary tubes have been made, depending upon the area to be treated. Medication has been greatly facilitated by the removal of pus and necrotic debris, especially in cavities as are found about the nose, throat, ear, and accessory sinuses, which are ordinarily not easily reached. Modifications of the various types of sinus probes and sounds have been devised so as to facilitate the cleansing of various sinuses.

After much experimentation with various suction handles for office use, we have finally devised one, we believe, which supports those principles which have bothered us considerably in the use of suction in the past (Fig. 1). This handle is moulded so as to fit conveniently in the hand with the cutoff worked very easily by the thumb. The angle of 45° for the attachment of the various tips makes a convenient angle for use in the nasal chambers and ears, without the interference of the handle in the line of vision. The attachment for the various canulae is of the bayonet variety in each of the six metal canulae. Included in this set are one short canula for the ear; a long, large lumen canula; one for the frontal sinus; one for the natural opening of the antrum; an antrum trochar; a long canula and trochar with a blunt end for the sphenoid. This handle and the tips are applicable for irrigation purposes as well as suction.

One cannot overestimate the value of suction over manipulation, particularly in acute diseases about the head. If statistics could be made so complete as to include what procedure

was used during the acute infection, before the complication set in, it might be definitely demonstrated that much in the way of the sequelae might have been avoided by the use of this carefully planned mechanical procedure.

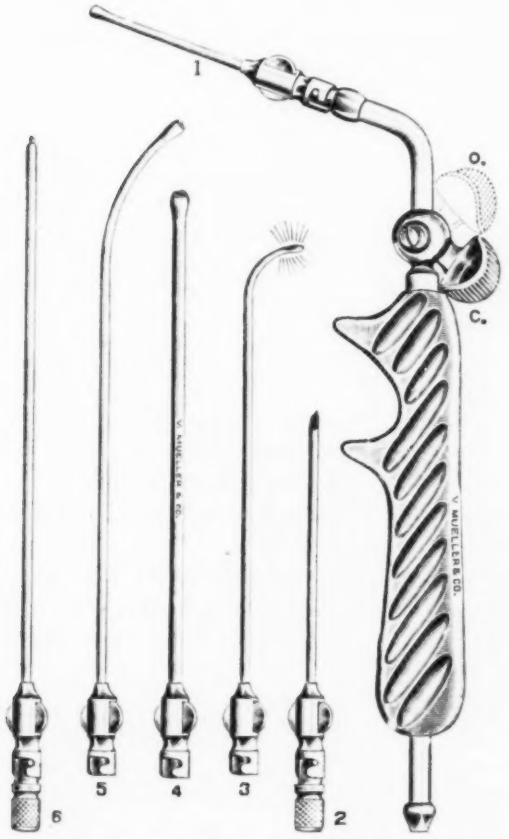


Fig. 1. Suction and Irrigation Handle and Canulae. Handle designed to fit comfortably in the hand and the cutoff easily controlled by the thumb. The bayonet attachment for the canulae is placed at a 45° angle so as to facilitate working in cavities without the handle interfering in the line of vision. There are six canulae included in the unit; (1) ear, (2) antrum trochar, (3) natural opening of antrum, (4) large lumen, (5) frontal sinus, (6) sphenoid (blunt pointed).

CAUTERY HANDLE AND TIPS.

Jos. C. Beck, M.D., Harry L. Pollock, M.D., and
Francis L. Lederer, M.D.

CHICAGO.

The use of the galvanocautery for cauterization in the upper straits of the nose has been abandoned for years because of the complications arising from its use. However, realizing the heresy of such an act, one of our group (F.L.L.) has employed it extensively in over fifty instances of the strictly polypoid cases, where radical removal of the middle turbinates and thorough exenteration and reexenteration of the ethmoid cells has failed to check the recurrence of polyps. These cases are purely those of the nonsuppurative, hyperplastic variety. Cases of meningitis following cauterizing of the middle turbinate (thrombosis of the ethmoidal veins) which have been reported in the literature, were performed under different circumstances than ours, namely, in the presence of chronic suppuration. Therefore, after careful consideration, our observations indicate that many sequelae and complications which have been ascribed to this form of procedure are due to entirely different causes, and, at the most, this procedure may have had only a determining influence.

The cautery unit presented here has helped solve the matter of technic considerably (Fig. 1). The make and break of the current are easily controlled, much on the order of a syringe. With the aid of Mr. George Wallerich, of V. Mueller & Company, we have succeeded in solving a definite problem in the cautery tips, in that they are bent so as to make a 45° angle with the handle. This enables one to work in the upper straits of the nose without having the handle interfere in the line of vision. These three cautery tips are made to serve the purpose of working in the upper straits of the nose (A); toward the lateral wall on the left (B); and the right side (C) of the nose and in the ethmoid regions.

We feel, that by the improved technic and the careful selection of cases, this method may come into use again.

However, being fearful lest its indiscriminate use should lead to mishaps which would again throw this method into discard, we are working up to a larger series of cases and will subsequently present it in detailed form.

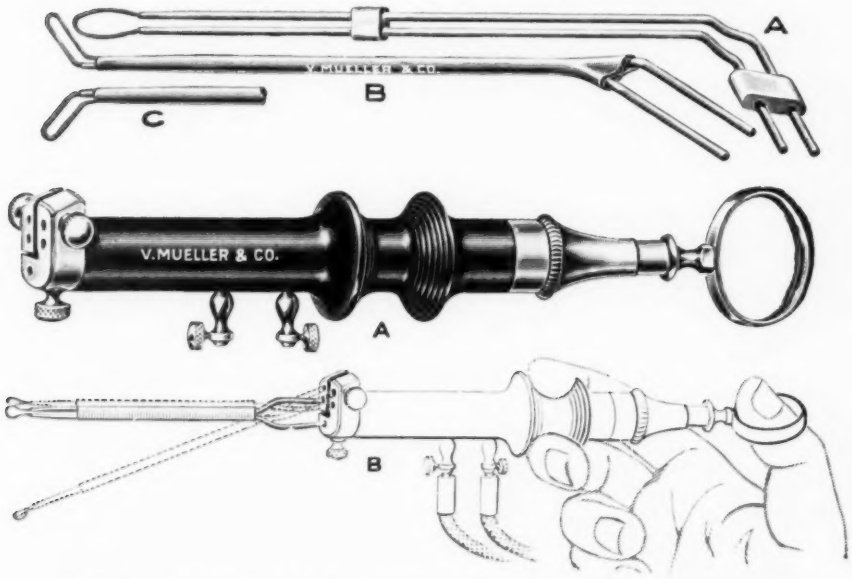


Fig. 1. Lederer-Wallerich Cautery Unit. A, B, C, Cautery tips. B illustrates angle of 45° for work in nose. The handle is applicable for all standard forms of cautery tips.

THE VISIBILITY OF THE RETINAL CAPILLARIES IN OPHTHALMOSCOPY WITH YELLOW-GREEN LIGHT.

JONAS S. FRIEDENWALD, M.D.

BALTIMORE, MD.

When Vogt devised his instrument for ophthalmoscopy with red free light[1], his main purpose was to discover whether or not the yellow color of the macula could be seen in the living. Only after this purpose had been fulfilled was it noticed by Vogt and others, that this light might be of use in clinical ophthalmoscopy. They discovered that the fundus could be seen in greater detail with this light than with ordinary white or yellow light. So far as I have been able to learn, this increase in visible detail has in general been attributed to the heightened contrast in the picture—the blood vessels, for instance, stand out sharp and black against the blue green background of the fundus. I believe this point has not received sufficient consideration, for I think that every one who has used the red free light of Vogt must realize that the ophthalmoscopic picture is not merely more contrastive, but more sharp and clear as well. This increase in the definition of the picture cannot wholly be explained on the basis of the increased contrast.

The human eye is in a sense a very poor optical instrument, its main defect being in its chromatic aberration, which is perfectly tremendous when compared with that of a good camera objective. There is about one diopter difference in the refractive power of the eye for light coming from opposite ends of the spectrum—an eye which is emmetropic for red light is one diopter myopic for violet light. Naturally, when we ophthalmoscope with mixed white light, the whole picture is slightly blurred and ill defined; outlines are soft and hazy, not harsh and clear. This is especially true, because the chromatic aberrations of the observed and observing eyes are additive. It must, then, be clear that the increase in definition noticed with the red free light is due to the fact, that this light

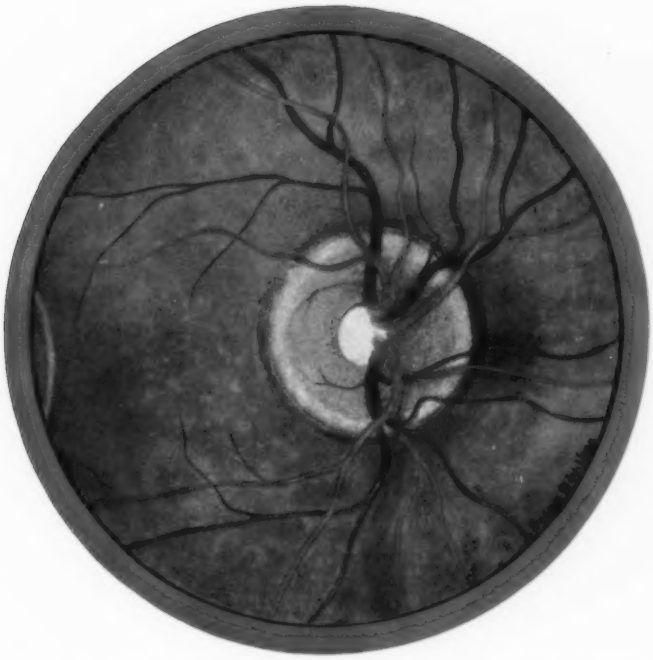


FIG. 1—NORMAL FUNDUS SEEN WITH ORDINARY ELECTRIC OPHTHALMOSCOPE.

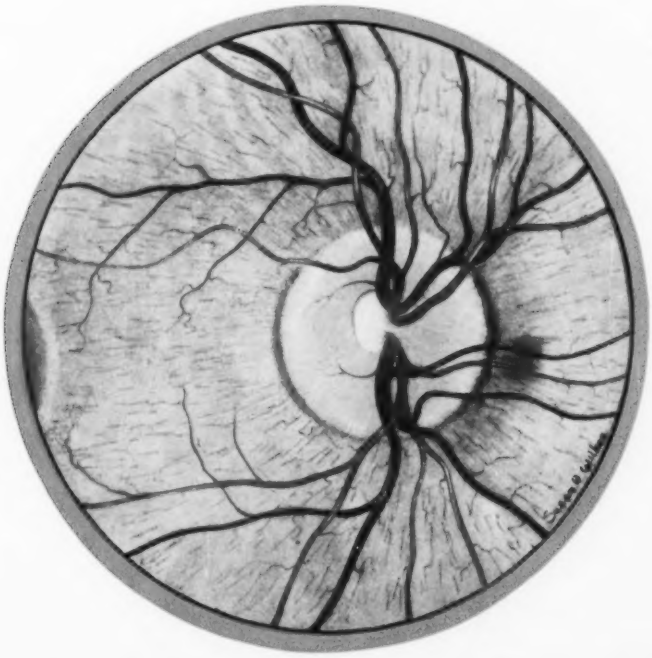


FIG. 2—SAME FUNDUS AS FIG. 1, SEEN WITH YELLOW GREEN LIGHT (FRIEDENWALD).

is of a more limited spectral range than the ordinary white or yellow light, and the aberrations in the image are correspondingly reduced.

In accordance with this theory, I have had constructed a simple treatment, a carbon arc with a solution of napthol green as light filter, (the detailed description will appear elsewhere)—the purpose being to produce a light of still more limited spectral range. This light is free from red and orange; it is also free from blue and violet; it contains only yellow and green light. The ophthalmoscopic picture obtained with this light is even sharper than that obtained with the red free light of Vogt.

A point of special interest is the fact, that in areas of the fundus which on ophthalmoscopy with white light appear quite free from blood vessels, we see innumerable fine dark

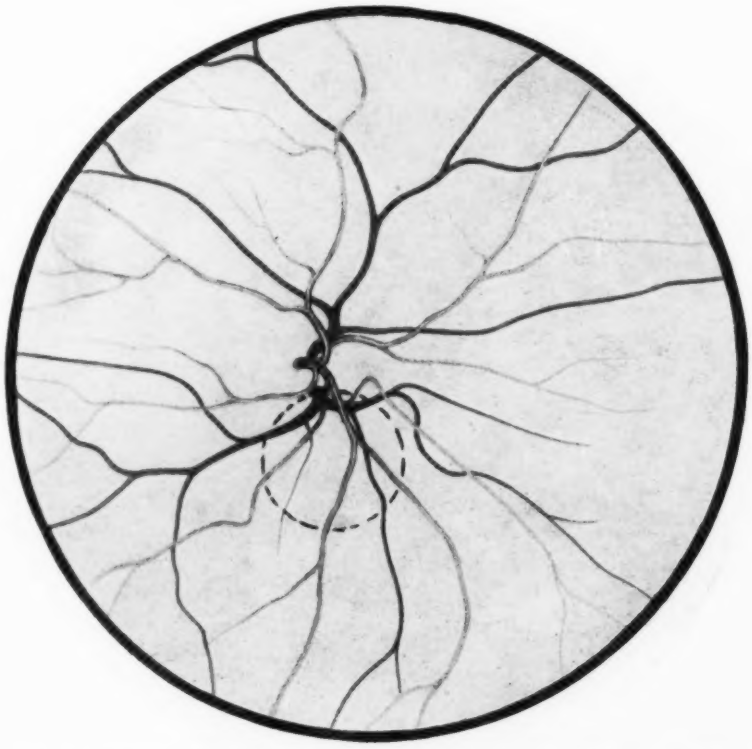


Fig. 3. Diagram of the retinal vessels shown by a postmortem injection of India ink. The area within the dotted circle is shown under higher magnification in Figs. 4 and 5.

lines, so fine as to be almost at the limit of vision, but, nevertheless, in this harsh illumination quite clearly visible. Some of these fine lines are seen to be connected with small arteries, some with small veins, some are seen in the intervening space, their connections with the remaining vascular tree being too fine to see.

In order to determine what order of vessels these were, I injected India ink into the blood vessels of the eyes of several cadavers. A study of these preparations makes it clear that the fine lines observed are capillaries; they correspond in distribution and in size relative to the larger vessels exactly to the capillaries seen in the injected specimens.

These injected specimens brought out a further point of interest which I do not believe has been previously noted, namely, that the capillaries of the retina form two clearly distinguishable nets. The more superficial net lies in the layer

Fig. 4. India ink injection of the retinal capillaries.

of nerve fibers, and the capillaries here tend to run in a direction parallel to that of the nerve fibers, with short connecting bridges running at right angles. The deeper layer is largely independent of the superficial one, connected with it only here and there by widely separated channels. It is much more irregular in pattern. It lies mainly in the inner nuclear layer of the retina, but sends occasional loops both up toward the layer of ganglion cells and down toward the nuclei of the rods and cones.

BIBLIOGRAPHY.

1. Vogt. Graefes Archiv. f. Ophth., 1913, vol. LXXXIV, p. 293.

Fig. 5. Same as Fig 4, with the superficial capillaries omitted. Not all of the capillaries are injected.

TRANSACTIONS

OF THE

TWENTY-NINTH ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

NECROLOGY

MINUTES

CONSTITUTION AND BY-LAWS

MEMBERSHIP ROLL

DEATHS

Dr. George Edgar Benson, Minneapolis, Minn.

Dr. Frederick N. Bigelow, Providence, R. I.

Dr. Horace Bonner, Dayton, Ohio.

Dr. James H. Crawford, Martin, Ga.

Dr. William S. Frost, Spokane, Wash.

Dr. Peter A. Haley, Charleston, W. Va.

Dr. Otis Orendorff, Canon City, Colo.

Dr. William Martin Reed, Kansas City, Mo.

Dr. Daniel Grant Sanor, Columbus, Ohio.

Dr. Horace Mann Starkey, Rockford, Ill.

Mr. Sydney Stephenson, London, Eng.

Dr. Hiram R. Stilwill, Denver, Colo.

Dr. O. C. Wolf, Columbus, Ohio.

MINUTES

September 16, 1924

The Twenty-ninth Annual Meeting of the American Academy of Ophthalmology and Oto-Laryngology was called to order at nine forty-five by the President, Dr. Walter B. Lancaster of Boston.

Following the Address of Welcome, it was moved by DR. A. J. BEDELL that the reading of the minutes be dispensed with. Motion seconded and carried.

DR. LUTHER C. PETER, Secretary, then read the following report:

SECRETARY'S REPORT

Mr. President and Fellows of the Academy: The Membership Roll now numbers 1,426. During the year, death has claimed twelve of our members. In this group of names is that of Mr. Sydney Stephenson of London, England, one of our former distinguished guests of honor and an Honorary Fellow of the Academy. Mr. Stephenson was a Fellow of the Ophthalmological Society of the United Kingdom, for many years Master of the Oxford Ophthalmological Congress, and Editor of the British Journal of Ophthalmology.

Many of our Fellows who practice the four specialties represented by the Academy have expressed the hope, that the program could be arranged so as to permit them to attend all of the sessions. The Program Committee, with the approval of the Council, has constructed the program for this year with this object in view. This plan has shortened the program to the extent of one session each. For future guidance in carrying out the wishes of the majority of the Fellows, the members of the Council will appreciate an expression of opinion as to which plan is preferred.

The Secretary offers his apologies for the tardiness in sending out the programs. Because of delinquency on the part of some of the readers of papers in forwarding their abstracts, the completed program did not reach the Secretary's

office until the 20th of August—five days after the time required by the By-Laws for the programs to be in the mail. By working overtime and far into the night, with an augmented force, the work of printing and mailing was accomplished in three days. For the benefit of the Program Committee which may function in the future, contributors to the program are earnestly required to assist the committee by promptness in forwarding their contributions. All material must be in the hands of the Secretary six weeks before the meeting in order to carry out the conditions of the By-Laws, which require the mailing of programs thirty days before the Annual Meeting.

The Secretary has called attention in the program to the many complaints which have reached his office of failure to secure the Transactions for the year. By a ruling of the Academy, only those who have paid their dues on or before March first are entitled to receive the Transactions free of charge. The mailing list of the Editor of those who are entitled to receive a copy of the Transactions is forwarded to him by the Treasurer. Because of the expense of printing, the Editor is authorized to have printed a very limited number of volumes in excess of the certified list supplied by the Treasurer. This year the supply was exhausted and many, after adjusting matters with the Treasurer, could not be supplied.

Changes in address should be forwarded promptly to the Secretary's office.

One hundred and twenty candidates have applied for admission this year. The list should be carefully studied by all Fellows, and any information as to the undesirability of any candidate should be communicated to an officer or a member of the Council. Only by this means is it possible to keep our membership roll free from undesirable or improperly qualified Fellows.

Respectfully submitted,

LUTHER C. PETER,

SECRETARY.

On motion, duly seconded, this report was accepted.

REPORT OF TREASURER

Balance on hand	$15,914.30	
Received from dues, int. on bonds, etc.	16,006.20	$31,920.50
Disbursements		8,712.83
Balance		$23,207.67
Cost of sixteen $1,000 bonds		15,959.77
Balance		$ 7,247.90
Previous bonds		$33,578.50
1924 bonds		15,959.77
Grand total		$56,786.17

SECORD H. LARGE,
Treasurer.

Moved by DR. L. C. PETER that this report be received and referred to the Auditing Committee. Motion seconded and carried.

REPORT OF COMMITTEE ON PUBLICITY AND SERVICE

The Committee begs to apologize for the delay in completing its work, and to assure the Academy that this has not been due to any lack of diligence on the part of the Committee. The matter was finally brought before each of the national associations interested in Ophthalmology and Oto-Laryngology, and favorably acted upon by each of them. They have appointed the following representatives on the National Council:

Section on Laryngology, A. M. A.....S. H. Large
Section on Ophthalmology, A. M. A.....W. R. Parker
American Laryngological Society.....Emil Mayer
American Otological Society.....T. J. Harris
Triological Society .....Ross H. Skillern
American Ophthalmological Society.....E. C. Ellett

It now remains for the Academy to appoint its representative, and the Committee suggests that the representative be an ophthalmologist, to balance the personnel of the committee between the specialties.

EMIL MAYER
ROSS H. SKILLERN
S. H. LARGE
H. W. LOEB
E. C. ELLETT

Moved by Dr. E. C. Ellett that this report be referred to the Council. Motion seconded and carried.

Dr. Harry S. Gradle, Chicago, made the following report for the Section on Instruction:

Report of Section on Instruction

The Section on Instruction presents its report in the form of literature that is sent out. The Committee has been extremely pleased with the attitude taken by the Academy members, both in the matter of attendance and in the matter of suggestions. It is realized by the Committee that its sole function is to serve the wishes of the members of the Academy in the kind of instruction course that is offered each year, and it is hoped by the Committee that the members will be free in their criticism of the courses as they are offered—a criticism that may be both destructive and constructive. Of course, it is impossible to follow all the suggestions offered, or to please everybody, but all the suggestions coming from members are weighed by the Committee, and it has endeavored to satisfy the requirements as they are expressed in the suggestions that have been offered. It is rather difficult to offer a course of instruction to as large and as varied a membership as the Academy possesses.

As far as a formal report goes, the Committe has nothing to offer, except that it is putting on a course that it hopes will satisfy you this year.

Harry S. Gradle.

This report was accepted.

Report of General Committee, Section on Ophthalmic and Oto-Laryngic Pathology

There has not been the response from the membership of the Academy and the profession at large to the Committee on Pathology that had been hoped. When the Committee, or rather, the Section on Pathology was first formed, the Committee pointed out that it hoped all of the men throughout the country who did not have facilities, either group or private laboratories, to carry out pathologic work would avail themselves of the Army Medical Museum. We have not published details beyond a few preliminary announcements that we sent out, and possibly there is still some doubt in the minds of the members as to exactly what the Section on Pathology can and does endeavor to accomplish.

Briefly, if any member of the Academy, or any physician, will send any pathologic material which he may obtain to the Army Medical Museum, Washington, D. C., accompanied by a history of the case from which the material was obtained, that material will be sectioned and studied by competent pathologists, and a report of the study and a diagnosis from the specimen will in due time be forwarded to the donor of the specimen. Furthermore, on request of the donor, cuts of the section or even pentographs of the section or gross material will be forwarded to the donor. This is entirely without cost to the donor of the specimen. The entire cost is borne by the Army Medical Museum, which gains by the acquisition of material that is beyond the possibilities of the Army. There is not sufficient ear, nose and throat work in the Army to supply the Museum with the variety of specimens necessary for a complete pathologic museum, and therefore Major Coupal welcomes this affiliation with the Academy in order to complete the Museum. The Committee hoped that the members would respond and cooperate better than they have done. There are a few members who are very faithful in sending in the material they obtain. Unfortunately, this number is not large enough, and we trust that more and more members, as time goes on, will avail themselves of the opportunities offered by the Museum. When sufficient material has been sent to the Museum to really form a complete collection of pathologic material of the eye, as well as the ear, nose and throat, the Army Museum will then be in position to offer a display to the Academy that will be without parallel. As it is, the display we have set up this year is somewhat limited.

It would seem unnecessary to reiterate the method of forwarding specimens to the Army Medical Museum. Specimens of any and all sorts are gladly received from all, provided they are accompanied by a full clinical history and description of the case. The pathologic material had best be preserved for twenty-four hours in a 1 to 3% solution of formalin and then packed in layers of cotton in a wide mouthed bottle. The cotton should be freely moistened, not saturated, with the same solution of formalin. The bottle should be labeled with the name of the donor and the character of the specimen, packed in a mailing carton, and forwarded to THE ARMY

MEDICAL MUSEUM, Washington, D. C., Section on Ophthalmic Pathology. The clinical description of the case should be forwarded at the same time under separate cover. Upon receipt of the specimen, it will be photographed, embedded, and sectioned, after which it will be studied and described by the various ophthalmic pathologists connected with this Section. A description of the specimen, together with the pathologic diagnosis, will be sent to the donor as rapidly as possible; but do not forget that it takes upward of three months to embed, section, and describe an eyeball. Upon request of the donor, copies of the photographs and individual sections will be forwarded.

This is all done without any expense on the part of the donor of the specimen, who gains by having a careful study of pathologic material that only too frequently is consigned to the garbage can. The Army Medical Museum gains by receiving pathologic material for the Museum that under other circumstances would be impossible to accumulate within the Army proper. However, the Section on Ophthalmic Pathology can be of use to us only if we cooperate by sending to the Museum all material that comes within our grasp, and the sooner the American ophthalmologists realize this and come to the aid of the Museum as indicated, the sooner will we have a Museum of Ophthalmic Pathology, second to none in the world.

HARRY S. GRADLE.

This report was accepted.

REPORT OF COMMITTEE ON THE HARD OF HEARING

To the President and Fellows of the Academy of Ophthalmology and Oto-Laryngology: Your Committee on the Hard of Hearing appointed last year by our retiring President, Doctor Carmody, is able at this time only to report progress. We find after deliberate consideration of the task which the creation of such a committee implies, that there are involved several component problems demanding more or less detailed investigation. This requires more time, and the cooperation of similar committees of our other national societies which are chiefly interested.

Since our last annual meeting, in response to the request of this Committee, the American Laryngological, Rhinological and Otological Society has expanded its Committee on the Deaf Child by the addition of four members, and enlarged

its scope to include the problems of the deafened. At the present time, all four of our national societies have similar committees, somewhat interlocking in personnel. Your own Committee also has several members who are very actively interested in the American Federation of Associations for the Hard of Hearing. Thus we are assured of the fullest cooperation of the various agencies now engaged in a study of the problems of the deafened.

That your Committee may ultimately accomplish its work in keeping with the purposes and ideals of this Academy, we would request that it be continued.

Respectfully submitted,
HOWARD V. DUTROW
HAROLD HAYES
CHARLES W. RICHARDSON
GEO. L. TOBEY
CLAUDE F. UREN
HORACE NEWHART, CHAIRMAN

This report was accepted.

REPORT OF COMMITTEE FOR OPHTHALMIC EXAMINATIONS

We have held a number of examinations this year. They have been rather better attended, and the quality of the work has been distinctly better. It is evident that the main object that led to the formation of this Board, namely, the elevation of the standards of teaching, is being attained. Men are better trained, unquestionably, and this is very gratifying to those who have had that in view. The number who have been certificated is close to 500, and I wish those who are not clear in their minds as to the method of procedure in getting this certificate would not hesitate to write the Secretary for the information they need. It is a simple matter to apply for a certificate, and it is a comparatively easy matter to pass all the requirements. The main requirements are those of good training and good character, and examination in the practical and theoretic sides of the subject.

WALTER B. LANCASTER.

This report was accepted.

REPORT OF COMMITTEE FOR EXAMINATION IN OTO-LARYNGOLOGY

A meeting of the Board of Examiners of this Academy was held at the Royal Victoria Hospital, Montreal, Canada, on September 15, 1924, at nine a. m., the following members being present: Drs. Beck, Carmody, Mosher and Skillern.

The following candidates were then examined.

Dr. Allen R. Cunningham.....Halifax, Nova Scotia
Dr. Ralph Almour.....New York City
Dr. W. A. McNichols.....Dixon, Illinois
Dr. John B. Morton.....Detroit, Michigan
Dr. Wm. C. Mussun.....Cleveland, Ohio
Dr. Lyman C. Richards.....Boston, Mass.
Dr. Walter D. Chase.....Bethlehem, Pa.
Dr. J. M. Croman.....Mt. Clemens, Michigan
Dr. Charles C. Jones.....Cincinnati, Ohio
Dr. Eugene E. Hinman.....Albany, N. Y.
Dr. Wm. F. Keim.....Newark, N. J.
Dr. David H. Ballon.....Montreal, Canada
Dr. Alfred A. Schwartz.....New York City
Dr. Paul Lux.....Kansas City, Missouri
Dr. F. E. Risdon.....Toronto, Canada
Dr. R. S. Pentecost.....Toronto, Canada
Dr. Walter H. Rieger.....Cleveland, Ohio
Dr. D. N. Cott.....Buffalo, N. Y.
Dr. Wm. P. Hoffman.....Davenport, Iowa
Dr. James A. Babbitt.....Philadelphia, Pa.
Dr. Robert H. Frazer.....Battle Creek, Michigan
Dr. A. E. Messier.....Worcester, Mass.

All these candidates, with the exception of Dr. Messier, fulfilled all requirements and are hereby recommended for admission to the Academy.

On account of the dissolution of this Board, it was recommended that the examining fee paid by Dr. Noah Fox be returned to him with letter of explanation.

In pursuance to certain communications, the National societies pertaining to laryngology, rhinology and otology appointed the following men to represent them in a committee to be known as the National Board of Examiners in Oto-Laryngology:

Section on Laryngology, A. M. A.	Drs. Beck and Lynch
American Otological	Drs. Loeb and Halstead

American Triological	Drs. Shurly and Spencer
American Laryngological	Drs. Mosher and Skillern
American Academy	Drs. Carmody and Wherry

A preliminary meeting was held with Drs. Beck, Mosher, Carmody and Skillern present, and it was decided to organize permanently at the meeting of the Southern section of the Triological Society, to be held at Palm Beach in January, 1925. Dr. Skillern was delegated temporary secretary to notify the various members and arrange for the place of meeting.

JOSEPH C. BECK
THOMAS E. CARMODY
HARRIS P. MOSHER
ROSS H. SKILLERN

A special meeting of the Board of Examiners of this Academy was held at the Mount Royal Hotel, Montreal, Canada, September 16, 1924, at twelve. The following members were present: Drs. Beck, Carmody and Skillern.

The following candidates were then examined:

Dr. Wm. Fowler.....Detroit, Michigan
Dr. Norman D. Buchanan.....Peterboro, Ontario
Dr. Leverett DeV. Chipman.....St. Johns, N. B.
Dr. John D. Moore.....Bloomfield, N. J.
Dr. James A. Fisher.....Asbury Park, N. J.

All these candidates were found to be qualified for membership in the Academy.

JOSEPH C. BECK
THOMAS E. CARMODY
ROSS HALL SKILLERN, SEC'Y.

Moved by DR. EMIL MAYER that this report be accepted. Motion seconded and carried.

DR. THOMAS E. CARMODY, Chairman of the Committee on Necrology, read the list of departed members as published in the program, with the addition of the name of Doctor O. C. Wolf, Columbus, Ohio. In honor of the departed, the members stood in silence for one minute.

After the scientific session, the meeting adjourned.

September 17, 1924

The session was called to order at nine-thirty by the President, Dr. Walter B. Lancaster. This being a business session,

the first order was action on proposed amendments to the Constitution and By-Laws.

THE SECRETARY: Most of the proposed amendments to the Constitution and By-Laws are simply a matter of phraseology; others are material changes. There have been a number of amendments made from time to time, and the Council felt that it was fitting to gather these amendments together and incorporate them in well phrased language.

CONSTITUTION

Article 3, Section A, to be replaced by:

"A candidate for active Fellowship shall be a member in good standing in his or her local, county, or State society, and shall have been practicing medicine in the special branches of eye, ear, nose, or throat exclusively for at least one year. The candidate shall present the regular application blank of the Academy duly filled out (and) endorsed by two active Fellows of the Academy, at least one of whom shall be a resident of his own city or state (except when there are no members of this society in the state), and accompanied by the entrance fee of ten dollars ($10.00). Such application shall be filed with the Secretary at least thirty days before the Annual meeting. A list of the applicants shall be sent to the Fellows with the final notice of the Annual Meeting.

"An applicant for membership must be approved by the American Board for Ophthalmic Examinations, or by the Committee in Oto-Laryngology. When, however, an applicant for Fellowship fails to pass the examinations of the American Board for Ophthalmic Examinations, or of the Committee in Oto-Laryngology, he shall not be eligible for election until he has passed the Board before which he failed. If an applicant shall fail to qualify for Fellowship within three years after his application has been filed with the Secretary, he must reapply for Fellowship in order to be continued on the list of applicants.

"Applications shall be presented to the Council, and after its approval, shall be submitted to a general vote of the Academy at any annual meeting. A three-fourths affirmative vote shall constitute an election."

DR. GEORGE F. KEIPER, Lafayette, Indiana: I wish to propose an amendment to this amendment, that after the words "State Society" the word "and Fellow of the American Medical Association" shall be inserted. This, for the reason that

we should declare our allegiance to the great American Medical Association in this matter.

DR. ROBERT SCOTT LAMB: I do not believe that is necessary, in view of the fact that in most State societies, a man must be a member of the American Medical Association before he can be a member of the society.

DR. KEIPER: Membership in his State society makes a man a member of the American Medical Association, but he is not a contributing Fellow. The American Medical Association is supported by the Fellows who contribute $5.00 a year and receive the Journal. I think most of our members are Fellows of the American Medical Association, but a special society of this kind, striving as we do for such high ideals, should surely link itself up with the ideals of the American Medical Association, with its 90,000 members and its more than 51,000 Fellows.

DR. S. H. LARGE: If we do that we will cut out all of our Canadian members, and we cannot do that.

THE PRESIDENT: For Doctor Keiper's information, I would like to say that it is the custom of the Council to consider that matter very carefully, and to reject candidates who are not members of their State Society and therefore of the American Medical Association, unless there is some very good reason.

Doctor Keiper's amendment was lost.

Moved by DR. EMIL MAYER that the Article be adopted as read. Motion seconded and carried.

Article 4, relating to Councillors: To change the number of Councillors elected from the floor from "four" to "six", and the term of service from "two" to "three" years.

In the third clause to omit the words "for election."

DR. WALTER R. PARKER: After this proposed change was presented and the matter was given more careful consideration, the Council has decided to recommend the following change in Article 4 as printed.

At the present time the Council consists of the President, Vice-President, Treasurer and two Ex-Presidents, and four Fellows. The President presides at the meetings. He is on the Council for the first year and presides at its deliberations, and is often in a very awkward position. To make his duties more intelligent, it has been proposed that he sit in the Council one year, with power to vote, but that he shall not

preside at the meetings until the second year. In other words, we would have a President and President-elect, much as in the American Medical Association at the present time. Then, to get Councillors who have had some experience, it is proposed that three of the Ex-Presidents sit in Council, and that three other Fellows with them constitute the Council.

To make that change, I would move that we substitute for the proposed change in Article 4 the following:

Article 4, Officers: After the word "President" insert the words "and President-elect".

Article 4, paragraph 2, second sentence changed to read as follows:

"The Council shall be composed of the President, who shall be chairman; the President-elect; the first Vice-President; the Treasurer; the three preceding Ex-Presidents, and three active Fellows, one to be elected each year to serve for a period of three years."

Simply to carry out the machinery, add a new paragraph between 2 and 3, as follows:

"The President-elect shall attend the meetings of the Council with power to vote, and shall assume the duties of President when this office is vacated, either by the expiration of the term of the President or for any other reason."

The motion was seconded.

Dr. Robert Scott Lamb: There may be some who object to the idea of having a close corporation. Of course, we have great reliance on the personnel of that organization, but it is a question whether some members would not prefer to have more members from the general body.

The President: For the benefit of those who did not hear, Doctor Lamb suggests that this change sounds like making the thing a close corporation, as though the Fellows who are chosen as Presidents were not active Fellows like those who are chosen members of the Council. As a matter of fact, all these eight or nine Fellows are active. The difference is in the manner in which they are elected. The officer chosen with the greatest care is probably the President. He becomes most familiar with the doings of the Academy. After a year of service, he is of more value. The first year, as I can testify, when I came on duty I was green. I was not acquainted with the workings. I was in an awkward position. I was unable efficiently to carry on the work of presiding over the Coun-

cil. It seemed to me if my predecessor could have set at my elbow to support me, things would have gone better. After awhile, I found out how to do it. It seems to me perhaps it would be judicious to separate this proposed change into two parts—the President-elect is a new matter, and it appears to the Chair that it may perhaps not be germane to the general tenor of the recommendation made last year. Therefore I would suggest that this be divided into two parts, and the part pertaining to the President-elect be proposed now and acted on next year. The part changing the number of Councillors and the way they are elected is perfectly germane to the proposal made last year, and therefore can be acted upon today.

Dr. Emil Mayer: Do I understand that the President-elect has no duties? I submit it is entirely germane. We now decide upon the additions to the Council, and it is quite within the province of the Academy that that should be done now, without putting it off another year.

The President: The Chair would like the decision of the Academy on that point, and will, therefore, ask for an expression of opinion as to whether you wish to take this up now, or wait until next year. Those who are in favor of the whole matter as presented by Doctor Parker, say Aye. Carried.

Vote on the amendments of Doctor Parker. Carried.

Vote on the changes in Article 4, as amended. Carried.

The Secretary: With reference to the last clause, I move that we strike out the words "in the affirmative," as they are superfluous. Motion seconded and carried.

The Secretary: The next refers to the list of applicants. Article 5—to change the latter part of the clause so as to read: "also mailed to each Fellow at least ten (10) days prior to the annual meeting at which the vote is to be taken."

Moved by Dr. Lamb that this be adopted. Motion seconded.

Dr. A. J. Bedell: I believe that ten days is too short a time. May I ask why there is contemplated a change from thirty days?

The Secretary: There is no contemplated change. There was no provision before this for submitting the list of applicants to the membership. The program is supposed to go out thirty days before the Annual meeting. It often happens, as

this year, that some members neglect to send in their contributions, and the program may be late. The Council therefore suggested ten days as a suitable time limit.

Motion carried.

By-Laws

Article 3, relating to Elections: In the first clause, change "occur" to read "take place".

Motion to adopt carried.

Article 4, relating to Duties of Officers: Insert in first clause before "Council" the words "of the" and omit "the" before "Chairman".

Motion to adopt carried.

To add the following sentence. "He shall fill by appointment all vacancies occurring in the personnel of the Committees."

Motion to adopt carried.

To insert after duties of the Editor of the Transactions:

A. The Board of Councillors.

B. The American Board for Ophthalmic Examinations: "Three of the nine members of the American Board for Ophthalmic Examinations represent the Academy. According to the By-Laws of the Board, one member is elected each year to serve three years beginning the first of January next following the meeting at which he is elected. No member of the Board may serve more than six years continuously. Members shall be chosen in the same manner as the presiding officers of the Society which they represent.

"In accordance with this arrangement, one Fellow shall be elected each year on nomination by the Council, to serve for three years as a member of the American Board for Ophthalmic Examinations."

C. The Committee on Oto-Laryngology: "A Committee of five active Fellows of the Academy shall be appointed annually by the President, with the advice of the Council, to examine candidates in Oto-Laryngology. This Committee shall serve until the National Board in Oto-Laryngology is organized."

D. The Section on Instruction: "The Section on Instruction shall be directed by four active Fellows of the Academy, one of whom shall be elected each year to serve for a period of four years."

E. The Committees: To replace the present clause by

the following: "The following Standing Committees shall be appointed by the President: The Committee on Arrangements, the Committee on Exhibits, the Examining Committee in Oto-Laryngology and the Committees on Ophthalmic and Oto-Laryngic Pathology."

Moved by DR. THOMAS E. CARMODY that in section "C" and "E", and other places where necessary, the title "Committee on Oto-Laryngology" be changed to "National Board for Examinations in Oto-Laryngology." Motion seconded and carried.

Moved by DR. AUSTIN A. HAYDEN that the five clauses as amended be adopted. Carried.

Article 5, relating to Dues: In the second sentence, to omit "only" and to add "to December thirty-first following his election".

To insert after the second sentence: "A copy of the Transactions of the preceding meeting will be forwarded free of charge to all Fellows whose annual dues are paid before March first."

To replace "roster" by the word "roll" in the last sentence, and to omit the clause "and cannot be reinstated until he has paid all lapsed dues."

Motion to adopt carried.

Article 6. New By-Law: Reelection and Reinstatement: "A Fellow whose name has been dropped from the roll for nonpayment of dues as provided in Article 5 may be readmitted into Fellowship by formally applying for admission as a new member.

"A Fellow who resigns from Fellowship in the Academy may be reinstated at the discretion of the Council by conforming to the requirements of the Society, as provided in Article 3 of the Constitution."

Motion to adopt carried.

Article 7, relating to Order of Business: Omit under No. 7 "Report of Standing Committees", the clause "Membership Committee".

Insert after "Program":

"Committee on Instruction

American Board for Ophthalmic Examinations

National Board for Examinations in Oto-Laryngology."

Motion to adopt carried.

DR. GEORGE F. KEIPER: I move that these be adopted as a

whole, and that the Constitution and By-Laws be modified as amended and printed in the Transactions.

Motion second by Dr. Austin A. Hayden.

Dr. Emil Mayer: I move to amend, that wherever the words "Committee on Oto-Laryngology" occur, this be corrected to agree with the new title of this Committee.

Amendment accepted, and amended motion carried.

Report of Council

Dr. Walter R. Parker: As senior member of the Council I beg to make the following report: Officers for the ensuing year:

President	Horace Newhart	Minneapolis
1st Vice-Pres.	John M. Wheeler	New York
2nd Vice-Pres.	Frank Brawley	Chicago
3rd Vice-Pres.	W. H. Roberts	Pasadena
Treasurer	S. H. Large	Cleveland
Secretary	Luther C. Peter	Philadelphia
Editor	Clarence Loeb	Chicago
Member Council	S. Hanford McKee	Montreal
Member of Ophthalmic Board	Lee M. Francis	Buffalo
Official Delegate to Convention of English Speaking Ophthalmological Societies, to be held in London, July 14-17, 1925	Walter B. Lancaster	Boston

Place of Meeting for 1925, Chicago.

Time to be announced later.

The Council would further recommend that the following be made Life members of the Academy:

Edward J. Brown.....Minneapolis
Myles Standish .....Boston
Thomas M. Stewart.....Cincinnati
Emil Mayer.....New York City

This report was accepted, and it was moved by Dr. Allen Greenwood that the members named be elected to Life Membership. Motion seconded and carried.

Dr. Walter R. Parker: We have provisions in the By-Laws and Constitution for getting us in and making us behave

after we are in, but no provision for putting us out. The Council therefore would submit, for future consideration, the following change in the Constitution:

Article 3 at the present time is in one sentence, which refers to membership and need not be changed. Therefore we propose that the present Article 3 be designated as "Section 1," with no change, that the following be added to Article 3 as "Section 2".

"The name of any Fellow may be dropped from the list of members if, in the unanimous opinion of Council, it is for the best interests of the Academy to take such action. Appeal may be made from action of the Council to the Academy as a whole at any annual meeting by any Fellow whose name is thus dropped. A two-thirds vote of the members present and voting will be necessary to drop a man from the list of members."

THE PRESIDENT: This will be printed and acted on next year.

Following the Business Session the President presented Dr. George E. de Schweinitz.

DR. GEORGE E. DE SCHWEINITZ: Mr. President and Members of the Academy: The President has very kindly permitted me to say a word or two in regard to the Conference of English speaking ophthalmologists which is scheduled to take place in London in July, 1925. As you know, it was impossible to bring about an International Congress of Ophthalmology. I shall not take your time to detail the reasons —they are quite obvious. In its place, there is to take place the Conference to which I have made reference. As you know, Mr. Treacher Collins is the President of the Committee on organization, with such men as Lister, Parsons and Paton, and they have rapidly perfected the organization.

This will be about the type of program: Of course the usual beginning of the Conference, and then two general discussions, the first on the evolution and development of binocular vision, which will be opened by Doctor Keith, the great anatomist. It is hoped that from this continent others will join in that opening discussion. The second general discussion will be on investigations by means of the diaphragm and Gullstrand lamp, the socalled "living" microscopy of the eye. This will be opened by various members of the Ophthalmological Society of Great Britain, experts in

the use of the slit lamp. Of course, from the United States and Canada, others will be asked to join in this conference. Then there will be, naturally, a series of meetings such as occur in every congress of this character, where volunteer papers will be presented and discussed. These papers which are accepted by the Committee will be edited by Dr. John Parsons, who will have charge of the scientific program. Large preparations have been made to entertain those who come scientifically in the various hospitals, museums and institutions of learning in London, and preparations will be made to see that visitors are taken to Oxford, Cambridge, Edinburgh, the Midlands, and in general, to any place in England where ophthalmologic work is done, or where they may wish to go simply for the purpose of seeing the place. The usual social functions will be arranged for.

I have had the opportunity within the last few months of being present at a number of the conferences which have been organized for the purpose of carrying on this work to which I make reference. I think it is a very unusual opportunity. It is, so far as I know, the first time that a large conference shall gather together composed entirely of English speaking ophthalmologists, that is, those who come from Great Britain and her colonies and possessions, from Ireland, Canada, etc., and those from the United States and her possessions. It is not, to use a common expression, a mere gesture; it is the real thing.

Not only is interest attaching itself to this Conference from the ophthalmologic standpoint, but the Government is interested. I had the pleasure of having several conferences with the American Ambassador, Mr. Frank Kellogg, who has been willing to do everything he can from the standpoint of the Government's sympathetic interest.

Now, members of the Academy, it seems to me that it is not only a duty, but it should be a great pleasure to give to this Conference not merely sympathetic interest, but also a strong cooperative effort. You will realize that it is not intended for one moment that this Conference shall be given as something apart from the ophthalmologists of other countries, but simply to show what can be done by the English speaking ophthalmologists in an international organization. Moreover, there will be an opportunity, and in fact a special time has been appointed, to discuss a future International

Congress, and no less a person than Professor Van der Hoeve has promised to come to present that side of the question before this Conference, he himself coming, as it is proper he should come, from what was in time gone by a neutral nation.

Therefore I—and I think I am right in saying that for the moment I represent the Committee on Organization in London—make an appeal to the members of this great organization which represents the largest interests of ophthalmology in the United States and in the Provinces of Canada—I make an appeal that you shall take cognizance of this Conference, that you shall go as visitors, or as contributors if you feel so inclined. It is a great step in a forward direction, because not only is its function to develop what we have in ophthalmology in English speaking countries, and to present it for the consideration of those who attend; but it is also a great step forward in the possibility of opening international congresses in the future. In other words, it tends to bring us into international scientific sympathy. Therefore, both from the standpoint of science and of loyalty to the country from which you come and the language which you speak, from loyalty to our British and Irish friends—from all these standpoints and from that of a larger interest, I ask this membership here to take these few words of mine into their hearts if they will, into their thoughts, and to translate their feelings and their thoughts into an active cooperation, and as many of you as can go to this Conference, support it in every way possible, and let the world see what an English speaking gathering can do. I have no manner of doubt that the world will look with favor on it, and be glad that it has looked.

Dr. W. Gordon M. Byers, Montreal: I want to corroborate what has been so splendidly outlined by Doctor de Schweinitz. I know a large number of my Canadian confreres and colleagues intend to be present. I have a letter from Mr. Treacher Collins in which he says that those who wish to read papers or to take part in the discussions or to exhibit specimens in the museum, should communicate with the secretary of the Scientific Committee, Mr. R. Foster Moore, 91 Harley St., London. A very warm invitation is extended to everyone to take part.

Dr. Edward Jackson: Many of us were in Washington in 1922, and will remember the session at which the ophthal-

mologists of America practically pledged their support to an International Congress to be held in London. There started the movement that will result in the Convention of English speaking ophthalmologists next July. We were then back of the movement and I think I can speak for the body of our membership and say to Doctor de Schweinitz, Doctor Byers and Col. Kirkpatrick that we are still back of it, and that we will do all in our power to make it a success.

We can do this thing first, every one of us join the Convention and send in our membership fee of ten dollars. We will want the Transactions anyhow, and that can be done by every one of us now, and it will have more weight now, will give more encouragement to those who are going forward with the movement, than if it is done later. And further, we can and should now begin to lay plans. It will not be possible for all of us to be in London. We cannot now see what obstacles may be in the way, but the thing to do is to begin now by taking a membership and preparing to go if we can.

If I know the Academy, and I think I do, I think we can promise those who have given us the invitation that we will stand by our original pledge.

The President then introduced the following guests of the Academy: Dr. Judson Daland, Philadelphia; Dr. Adalbert Fuchs and Prof. Haslinger of Vienna.

September 18, 1294

The session was called to order at nine-thirty by the President.

Dr. Walter R. Parker: In compliance with the changed Constitution as adopted yesterday, I am instructed by the Council to make the following nomination:

For President-Elect, Dr. E. C. Ellett.....Memphis

To serve on the Section on Instruction for the remaining 4 years, Wm. L. Benedict.....Rochester

Under the head of Election of Officers, the list read at the Wednesday morning session, together with the two nominated this morning, was reread.

Dr. Emil Mayer: I move that the Secretary cast the ballot of the Academy for those named. Motion seconded and carried.

Dr. Horace Newhart, the newly elected President, was escorted to the platform and presided over the session.

Election of Fellows

The following candidates have been approved by the Council and have qualified in every respect:

Adams, Dr. Frank Mears.....Providence, R. I.
Babbitt, Dr. James Addison.....Philadelphia
Ballon, Dr. David Herbert.....Montreal
Bass, Dr. A. L.....Louisville, Ky.
Beard, Dr. Harold Jolly.....Youngstown, Ohio
Boone, Dr. James Lunsford.....New York
Bousquet, Dr. Joseph P. E.....Montreal
Chase, Dr. Walter Daniel.....Bethlehem, Penn.
Cook, Dr. W. Albert.....Tulsa, Okla.
Cott, Dr. Delzon N.....Buffalo
Croman, Dr. Joseph M., Jr.....Mt. Clemens, Mich.
Cunningham, Dr. Allan R.....Halifax, N. S.
Cushman, Dr. Beulah.....Chicago
Davidson, Dr. Herman P.....Chicago
Dunnington, Dr. John Hughes.....New York
Fairing, Dr. John Walker.....Greensburg, Penn.
Flynn, Dr. James Augustin.....Washington, D. C.
Glasford, Dr. Samuel Theodore.....Pekin, Ill.
Goodyear, Dr. Henry Marks.....Cincinnati, Ohio
Gracey, Dr. George Fay.....Harrisburg, Penn.
Hall, Dr. Alice K.....Chicago
Heath, Dr. Parker.....Detroit, Mich.
Higgins, Dr. Reuben Paul.....New York
Hinman, Dr. Eugene Emerson.....Albany, N. Y.
Hogan, Dr. Walter Louis.....Hartford, Conn.
Jones, Dr. Charles C.....Cincinnati, Ohio
Joynt, Dr. Albert J.....Waterloo, Ia.
Kahn, Dr. Myron Ellis.....Chicago
Keim, Dr. William Franklin.....Newark, N. J.
Kennon, Dr. William Gilliam.....Nashville, Tenn.
Kilbourne, Dr. Perley H.....Dayton, Ohio
Kirby, Dr. Daniel B.....New York
Lassalle, Dr. Albert.....Montreal
McDermott, Dr. John J.....St. Joseph, Mich.
Morton, Dr. John B.....Detroit, Mich.
Mussun, Dr. William George.....Cleveland, Ohio
Norcross, Dr. Edward P.....Chicago
O'Brien, Dr. Cecil Sterling.....Toledo, Ohio

Pearlman, Dr. Samuel James.....Chicago
Reuling, Dr. F. Harold.....Waterloo, Ia.
Rice, Dr. John Evarts.....Worcester, Mass.
Richards, Dr. Lyman.....Boston
Rieger, Dr. Walter Henry.....Cleveland, Ohio
Risdon, Dr. Ernest Fulton.....Toronto, Can.
Russell, Dr. Ralph E.....Waterloo, Ia.
Schurter, Dr. Maximillian Adolph.....Riverside, N. J.
Schuster, Dr. Stephen A.....El Paso, Tex.
Stein, Dr. Jules C.....Chicago
Vail, Dr. Harris Holmes.....Cincinnati, Ohio
Van Poole, Dr. Gideon McDonald.....Honolulu, Hawaii
Wold, Dr. Karl C.....St. Paul, Minn.

Moved by Dr. Emil Mayer that the Secretary cast the ballot of the Academy for the names read. Motion seconded and carried.

Report of Committee for the Standardization of Tuning Forks and Hearing Tests

As a result of a resolution passed by the Section on Oto-Laryngology of the American Academy at its meeting in Philadelphia, October, 1921, a committee for the standardization of tuning forks and hearing tests was appointed. This committee made a report at the meeting of the Academy in 1922, at Minneapolis, and again at the meeting held in Washington, in October, 1923. In order to familiarize the members with these reports and refresh their memory, we will give a summary of some of the previous statements. In our last report, we have a resume of the recommendations of the Committee of the American Otological Society, whose Chairman, Dr. Edward B. Dench, very kindly gave us their conclusions. This Committee adopted for testing the low limit the Dench tuning fork of from 26 to 64 double vibrations, and a fork of 256 double vibrations for bone conduction, the latter being made with a broad base so as to be applied more easily to the mastoid. The committee further stated that investigation was being undertaken regarding the standardization of the Galton whistle, but it had been found impossible to find a maker for the standard Galton whistle except the Edelmann, thus making the instrument very expensive. The committee then considered the possibility of having only a

part of the scale standardized, namely, 6, 12, 18 and 24,000 double vibrations so as to determine the upper tone limit, but the manufacturers who were approached did not consider this plan feasible as yet. The committee also stated that the Galton whistle at present on the market is not accurate, and that an approximate estimate may be made recording the upper tone limit in cases as "being slightly reduced, moderately reduced, much reduced, and greatly reduced, corresponding to 24, 18, 12 and 6,000 double vibrations." We are at this time presenting to you a report which covers a resume of the previous ones, together with such modifications and additions as seem advisable to your committee.

First: With reference to the character of the fork to be employed. This should be well balanced, of rustless metal if such can be obtained (oxidized steel perhaps will be suitable), of definite pitch, with definite duration of vibration, moderate in price and so made as to eliminate loud overtones, preferably by means of weights on the lower pitched forks, and of certain standard dimensions so that these can be made universal. To avoid rattling of the weights, one manufacturer uses round, flat clamp like devices in place of the projecting, easily broken thumb screws. This mechanism, however, is patented and therefore cannot be used by other manufacturers without the payment of royalty.

Second: The fork should be held uniformly near the ear, preferably with the wide surface of the prongs near the ear, without touching either the auricle or the vibrissae, in order to get the maximum sound wave and to avoid interference waves which occur at certain points, especially at the angle between the broad and narrow surfaces, holding the lower third near the ear to get the percussion point area. One of the members of your committee (Sonnenschein) has shown by tests with the phonometer and otherwise, that from the broad surface of the prongs the greatest intensity of sound is emitted.

Third: There is no unanimity in the committee as yet regarding the method of stimulating the fork or of methods of timing the fork. It has not definitely been decided whether it is better to note the duration of time that the fork is heard, or to describe the degree of hearing by the distance from the ear at which the fork can be appreciated, using either a control or knowing the duration of the fork's vibration. How-

ever, it seems that neither method is wholly accurate. Both of them should be tried out for some time before a definite conclusion is reached. To get uniform results, there should be a uniform method of excitation of the fork. If the examiner has defective hearing, he should use a normal control. At about the junction of the upper and middle third of the prong of the fork, there is what is known as the "percussion point," where there is the greatest intensity of sound and the least overtones. It would therefore be advisable to strike the fork at this place. With the lowest forks, such as those of from 16 to 36 double vibrations, excitation may best be made by allowing the fork to fall of its own weight, so that the center of percussion strikes upon the soft part of the thigh, the ball of the hand, or the knee. This may also be done with forks up to 128 double vibrations, by striking them lightly against the ball of the palm of the hand, care being taken to withdraw them from the object which they strike as quickly as possible, in order to prevent damping by a second contact. Some investigators claim that it is difficult if not impossible to get uniform results by striking the fork against the thigh. This point should be investigated further. In place of this method, a rubber pleximeter or brass rod can be used for actuating the forks. Forks up to c^3 may be excited by striking them with a light firm tap against the nail of the middle or other finger, care being taken that there is no damping by a second contact. For the c^4 and c^5 (respectively 2048 and 4096 vibrations), different degrees of excitation may be used, depending upon the acuity of hearing of the patient. The lightest excitation is rubbing the prongs between two of the fingers. If the sound is then not heard, strike one of the prongs against one of the finger nails; and if this is not appreciated, strike the broad surface of one of the prongs with a metal hammer. In presenting these suggestions for the excitation of the various forks, it should be noted that for the lowest ones soft objects like the hand or thigh or a rubber pleximeter should be used, and for the very highest tones, a metal hammer; in other words, the lower pitched the fork, the softer should be the object which strikes them.

Fourth: The committee recommends that at first only three standard tests be considered, namely, the Weber, the Schwabach and the Rinne. For the Weber test alone, a fairly low pitched fork from 100 to 200 double vibrations is recom-

mended. When the Schwabach is performed by placing the fork on the median line of the vertex or forehead, a similar fork, fairly heavy, so as to rest of its own weight, is desirable. For the Rinne (including the Schwabach when the bone conduction factor of the Rinne is used for this purpose) a medium pitched fork of 150 to 435 double vibrations should be used. The time during which the fork is heard by bone and air conduction should be definitely designated. The construction of the fork should be such, that the difference between the air and bone conduction is about 30 to 40 seconds. When vibrating in the air alone, the fork should be heard approximately 100 seconds. This, of course, requires a rather expensive fork. The upper limits of hearing can further be tested by means of the Galton (or Edelmann-Galton) whistle or the monochord. The latter is the only instrument whereby high tones can be tested by bone conduction.

Regarding the forks to be used, the committee recommends that at least five forks, (namely, C 32, c 128, c^1 256, c^2 512, and c^3 4096 double vibrations per second) be secured by those doing functional testing. It has been suggested to the committee, that if we would decide definitely on the forks we should all use and if this idea were agreeable to the members in general, it might be possible to design forks of special shape and weight or perhaps have a special name. This design could perhaps be patented or the name copyrighted, and the patent or copyright owned by the Society, which in turn could grant permission to any reputable manufacturing concern to make this particular fork or forks. This would not be a drain upon those buying them, and would add a certain amount of stimulus to purchasers of forks, besides bringing about the much desired uniformity in tuning forks employed for ordinary tests.

Although a number of efforts have been made, we have as yet been unable to find any manufacturer who can make forks of a rustless metal that will give as good a quality of tone and duration of vibration as the steel forks at present in use. Whether oxidized steel or a special rustless metal will be feasible remains to be seen. However, we believe that further efforts should be made in this direction, since rustless forks would be of a great advantage in many ways. If a fork is nickled and the nickling later on peels, we get adventitious sounds from the loose pieces of metal. On the other hand, if

the forks are unnickled and are not properly oiled or otherwise protected from the air, rusting usually takes place.

Some of your committee are in favor of using only the c forks. Others have been accustomed to employing the Bezold A (108 double vibrations) for the Weber and Schwabach tests, and a^1 (435 double vibrations) for the Rinne test. Most of these forks have advantages hard to find in some of the other forks, but nevertheless they are not essential; and even if others are employed, the same result will be obtained if the same care is taken in making the tests as outlined by Bezold in using the forks mentioned.

With reference to the recording of tuning fork and other functional tests, some members of your committee have for some time employed the acoumetric formula adopted at the International Congress at Buda Pesth in 1909, and have found it useful and compact. Others use the system of the Vienna Clinic, the diagram being stamped on the card and the findings noted thereon. There are others who measure the distance at which forks are heard, and express the degree of hearing in fractions, the denominator of which is the distance at which that particular fork is heard by the normal ear, and the numerator, the distance heard by the patient who is being examined. Both of these methods are fairly accurate if done carefully and systematically but neither of them is perfect.

SUMMARY: The committee recommends first: A list of five forks to be used in ordinary work, 32, 128, 256, 512 and 4096 double vibrations. In actuating these forks, the lowest is struck either against the ball of the palm of the hand or the fleshy portion of the thigh or actuated by means of a rubber plexímeter. The highest forks may be excited in various ways, depending upon the intensity desired, either by rubbing the prongs with the fingers, striking with the finger nails, or with a metal hammer.

Second: For the highest tones, either an Edelmann-Galton whistle or the monochord should be employed.

Third: An effort should be made to investigate the feasibility of the employment of a rustless metal for the manufacture of forks.

Fourth: Further study should be made of the methods of the recording of tests and the conduction, if possible, of experiments with reference to distance and timing of forks in functional testing.

Finally: We suggest that the committee be continued for another year in an effort to further solve some of the problems outlined.

Respectfully submitted,
L. W. DEAN
G. W. MACKENZIE
E. G. GILL
ROBERT SONNENSCHEIN, CHAIRMAN.

Moved by DR. WALTER R. PARKER that the report be accepted and the committee continued. Motion seconded and carried.

DR. ROSS H. SKILLERN: I have an announcement to make regarding the new Board for Examinations in Oto-Laryngology. There seems to be some misunderstanding regarding the exact title and the functions of this Board. The Board will be called the National Board of Examiners in Oto-Laryngology, and consists of ten members, two from each of the national societies in oto-laryngology. These men have already been appointed, and will meet at Palm Beach some time in January for the purpose of organization. Details will be worked out at that time. It is proposed however, that this Board examine all candidates for admission into the various societies. It will act in exactly the same way as the American Board for Ophthalmic Examinations. Certificates will be given, and the men will enjoy all the rights and privileges that certificates give those in ophthalmology. The Board will function at the meeting of the American Medical Association next summer.

DR. EMIL MAYER: I would like to present the following resolution:

"RESOLVED: That the former method of arranging the program of the Academy, in having the sessions of the ophthalmologic and otologic divisions take place at the same time in appropriate halls be adhered to."

In presenting this resolution there is not intended any criticism of the present program or committee, but believing that the best interests of the future scientific work of the Academy are involved, and as an expression of opinion of the members on the present arrangement has been requested, I respectfully submit my own views for your consideration.

For 28 years the Academy has adhered to the old form of joint meetings followed by sessions in two halls. The won-

derful growth of the Academy seems to me an endorsement of the value of the scientific sessions as formerly held.

As now arranged, there is no reason why members solely interested in one branch should not conclude that their interest in the scientific session has ceased, and return to their homes at noon of the third day. Formerly they remained until the full three days' sessions were completed.

The present arrangement places the Academy in line with State societies, and is, in the opinion of the speaker, a decided detraction from the great value of the Academy as a powerful educator, and a distinct influence in American medicine. It is an entering wedge of division in our ranks, that may result in lessening the value of the Academy as a factor among special organizations.

While it is true that, with a double program, one may now and then miss a paper that appears at the same time as another, the present arrangement will undoubtedly result in a diminished attendance on the part of readers of papers during the entire session. The result will be that readers of papers will appear only for the hour arranged for them, instead of being constantly present and lending by their participation in the discussions a great value to the proceedings.

In the interests of the Academy I sincerely trust that the motion will prevail. I move its adoption. Motion seconded.

THE PRESIDENT: I would like Doctor Parker to tell us the views of Council in this regard, as he was a member at the time it was introduced.

DR. WALTER R. PARKER: The change in the program as carried out this year was suggested by those who were interested in the four specialties covered by the Academy—those doing eye, ear, nose and throat work—who were frequently in one meeting when a paper in which they were interested was being presented in another section. It was solely to meet this demand that this question was taken up. It seems to me purely an arrangement that will accommodate the greatest number of the members. We have no argument as a Council, we have no scheme of our own to put forward. Whether it goes on this way or back to the old plan rests with the majority of the members. We have only one suggestion to make. We have not tried the plan long enough to know that it does not work. As a scientific body, I think we feel when we set out to do a piece of work, we should not change

our course until we know we are wrong. It is purely a question, as far as Council is concerned, of the convenience of the majority of the members. We have no argument one way or the other.

DR. WILLIAM H. SEARS. As one of the members who has something to do with all four of the specialties mentioned, we know there are a number of men who attend the meeting of the American Academy who do not get to any other meeting during the year, and those men have said to me that they were delighted with this arrangement of the program. If there was any other way by which they would have access to all the papers that are being given it would answer just as well, for instance, with the addition of another day. But these men are anxious to get everything they can during the days of the convention.

DR. GEORGE F. KEIPER: This present arrangement is nothing new or novel. The American Medical Association is following this plan, as you know, the Section on Ophthalmology meeting in the morning, and the Section on Oto-Laryngology in the afternoon, and vice versa. It may be that it is not wise at this juncture to take a vote on this proposition, and I will throw out the suggestion that it might be a good idea to have a referendum in order that all the members of the Academy may have an opportunity to vote, because at the present time I believe there is only about 10 per cent of the membership in this hall. I move, as an amendment, that this matter be made a referendum, and vote thereby taken of the entire membership of the Academy.

Amendment accepted by Dr. Mayer.

Moved, as a further amendment, that after this present plan has been tried for another year, the matter be considered by referendum. Amendment accepted and the amended motion carried.

DR. E. C. ELLETT: I have a matter to bring up here which has nothing to do with the specialties of ophthalmology and oto-laryngology, but which has a great deal to do with the medical profession, and it is something that I think should be brought to the attention of every medical society in this country.

In paragraph 10, Section 202, of public document 242 of the 68th Congress, being the World War Federal Act 24, approved June 7, 1924, on page 16 the following appears:

"The Director (of the Veteran Bureau) is further authorized, so far as he shall find Government facilities permit, to furnish hospitalization and necessary travelling expenses to veterans of any war, military occupation or military expedition since 1897, not dishonorably discharged, without regard to the nature or origin of their disabilities."

This means that any exservice man can secure free hospitalization and medical treatment from the Government for any illness or injury that befalls him. The question of its relation to his service is not to be raised. Any acute illness or any injury, not excepting venereal disease, entitles him to transportation at Government expense to a hospital, where he is cared for indefinitely, or until cured. That is communism, State medicine, and sovietism, all mixed into one. It tends to pauperize these four million men who were in the service of the Government during the war, and the injustice to the medical profession, especially those who are coming on, the future doctors, is tremendous; and the burden of taxation that it will ultimately result in is incalculable.

This thing is a law. It is not generally known, so the men who are entitled to its benefits are not beginning to apply for them. I think it would be very fitting that this organization, all of whose men did their duty to the Government during the war, should be the first to try to get this matter repealed.

I move that this question be referred to the Council for their consideration and advice, and I would be very glad to know if this thing impresses some of the other members as it does me.

Motion seconded and carried.

Dr. David W. Wells, Boston: I should like to offer the following motion:

"Since the rate of development of any cataract is always uncertain, and since some never increase and in some cases the lens becomes clearer, in order to determine the value of any therapeutic method, one should be able to compare any series of results of a special treatment with an equal number of similar cases untreated, and as such data are not now available, I move that the expediency of appointing a committee to study the natural history of untreated senile cataract be referred to the Council, with full power to act."

Motion seconded and carried.

Dr. George F. Keiper: The development of ophthalmology and oto-laryngology rests upon us first, as individuals, and secondly, associated together. It has seemed to some that the time has pretty nearly arrived when we should have State units of this Academy. We already have in a great many States our academies and sections of State societies, all of which indicate this general trend. I move that the matter of consideration of State units be referred to the Council, so that we may have more frequent meetings of our State members, after the manner of the American College of Physicians and Surgeons.

Motion seconded, but lost.

Chicago as the place of meeting for 1925 was approved.

Dr. Walter R. Parker: Before the Business Session closes, as Chairman and senior member of Council, it is the greatest pleasure of my office at this time to express to Doctor McKee and his Committee of Arrangements our sincere appreciation of all of the work they have done, and of the perfection of their arrangements. In this connection I wish to mention the care they have taken of our ladies. And last, but not least, I am sure you will say that last night we had one of the best evenings we have ever had in the Academy. Doctor McKee has our sincere thanks and appreciation, which I hope he will communicate to his coworkers. Rising vote of appreciation given.

Meeting adjourned.

CONSTITUTION

ARTICLE 1

NAME

The name of this society shall be THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

ARTICLE 2

OBJECT

The object of this Academy shall be to promote and advance the science and art of medicine appertaining to the eye, ear, nose and throat; and to encourage the study of the relationship of these specialties to surgery, general medicine and hygiene.

ARTICLE 3

FELLOWSHIP

The membership of this Academy shall consist of ACTIVE FELLOWS AND HONORARY FELLOWS, and their qualifications shall be as follows:

(a) A candidate for ACTIVE FELLOWSHIP shall be a member in good standing in his or her local, county, or state society, and shall have been practicing medicine in the special branches of eye, ear, nose, or throat exclusively for at least one year. The candidate shall present the regular application blank of the Academy duly filled out (and) indorsed by two Active Fellows of the Academy, at least one of whom shall be a resident of his own city or state (except when there are no members of this society in the state), and accompanied by the entrance fee of ten dollars ($10.00). Such application shall be filed with the Secretary at least thirty days before the annual meeting. A list of the applicants shall be sent to the Fellows with the final notice of the annual meeting.

An applicant for membership must be approved by the American Board for Ophthalmic Examinations, or by the National Board of Examiners in Oto-Laryngology. When

however, an applicant for FELLOWSHIP fails to pass the examinations of the American Board for Ophthalmic Examinations or of the National Board of Examiners in Oto-Laryngology, he shall not be eligible for election until he has passed the Board before which he failed. If an applicant shall fail to qualify for FELLOWSHIP within three years after his application has been filed with the Secretary, he must reapply for FELLOWSHIP in order to be continued on the list of applicants.

Applications shall be presented to the Council and, after its approval, shall be submitted to a general vote of the Academy at any annual meeting. A three-fourths affirmative vote shall constitute an election.

(b) Any Doctor of Medicine who has materially aided the progress of medicine in its relation to diseases of the eye, ear, nose or throat, may, on nomination by two Fellows and recommendation of the Council, and by a unanimous vote of the Fellows present at an annual meeting, be elected an ACTIVE FELLOW.

(c) Anyone who has signally aided the progress of medicine in its relation to diseases of the eye, ear, nose or throat, may, on recommendation of the Council, and by a unanimous vote of the Academy at any annual meeting, be elected an HONORARY MEMBER.

(d) The Council, for sufficient reasons, may recommend members to LIFE MEMBERSHIP without dues, and a unanimous vote of the members present shall constitute an election.

ARTICLE 4

OFFICERS

The Officers of this Academy shall be a PRESIDENT, a PRESIDENT-ELECT, FIRST VICE-PRESIDENT, SECOND VICE-PRESIDENT, THIRD VICE-PRESIDENT, SECRETARY, TREASURER, EDITOR OF THE TRANSACTIONS, and a BOARD OF COUNCILLORS.

THE PRESIDENT, PRESIDENT-ELECT, VICE-PRESIDENTS, SECRETARY, TREASURER, EDITOR OF THE TRANSACTIONS, and the new member of the Council shall be elected annually at a regular meeting. The COUNCIL shall be composed of the PRESIDENT, who shall be Chairman; the PRESIDENT-ELECT; the FIRST VICE-PRESIDENT; the TREASURER; the three preceding EX-PRESIDENTS; and three ACTIVE FELLOWS, one to be elected each year to serve for a period of three years.

Nominations for Officers are presented to the Academy by the Council. Other nominations may be made by a written petition signed by five (5) Active Fellows of the Academy.

The nominations shall be made and election held at an annual meeting. A majority vote cast by the Active Fellows present at such meeting shall constitute an election.

ARTICLE 5

AMENDMENTS

This Constitution may be amended by a two-thirds vote of the Active Fellows present at any annual meeting, notice of the proposed amendment having been given at the previous annual meeting, and also mailed to each Fellow at least ten (10) days prior to the annual meeting at which the vote is to be taken.

BY-LAWS

ARTICLE 1

ANNUAL MEETINGS

The ANNUAL MEETING shall convene at such time as the Council may recommend. The place of meeting shall be decided by the Council, notice of which shall be given to each member at least three months in advance.

ARTICLE 2

PAPERS

Twenty minutes shall be the maximum time allowed for the reading of a paper. The Fellow designated to open the discussion shall be allowed ten minutes. In the general discussion, no one shall speak more than five minutes, nor shall he discuss any paper more than once. All such papers shall be edited by the Editor of the Transactions.

No member shall read a paper that has been previously read or published.

All papers read before the Academy shall immediately become the property of the Academy, and must be delivered to the Secretary at the conclusion of their reading. All such papers shall be edited by the Editor of the Transactions, and their publication in the Transactions shall be under the control of the Council.

Every paper must be read in person by its author, unless good and valid reasons for his absence can be shown, when, upon a unanimous vote of the Fellows present, the paper may be read by proxy.

ARTICLE 3

ELECTIONS

The nomination and election of Officers shall take place at the annual meeting.

Only ACTIVE FELLOWS in good standing are eligible to hold office, and to vote on matters properly belonging to the Executive Session.

ARTICLE 4

DUTIES OF OFFICERS, ETC.

THE PRESIDENT

The PRESIDENT shall preside at all meetings of the Academy and of the Council, and shall act as the executive officer of the Academy, and shall be Ex-Officio Chairman of the Council and Chairman of the Program Committee. He shall fill by appointment all vacancies occurring in the personnel of the committees.

THE PRESIDENT-ELECT

The PRESIDENT-ELECT shall attend the meetings of the Council with power to vote, and shall assume the duties of President when this office is vacated, either by the expiration of the term of the President or for any other reason.

THE VICE-PRESIDENTS

The VICE-PRESIDENTS, in order of seniority, in the absence of the President, shall perform the duties of the President.

THE SECRETARY

The SECRETARY shall keep a record of the Academy meetings, as well as of the Council meetings, of which he shall be ex-officio clerk.

He shall superintend the publication of the Transactions and otherwise assist the Editor of the Transactions.

He shall assist the president in preparing the annual scientific program, and attend to its publication and distribution.

He shall receive an honorarium of two hundred dollars a year for his services.

The Treasurer

The Treasurer shall take charge of all moneys of the Academy; keep an account of the same; pay all bills approved by the Council and signed by the President and the Secretary, and he shall present a statement of the same to the Academy at its annual meeting.

He shall be under bond for twice the amount of funds ordinarily carried as surplus, the cost of the bond to be paid for by the Academy.

He shall keep a correct list of the members who have paid their dues, and notify the Secretary of all delinquents.

The Editor of the Transactions

The Editor of the Transactions shall receive all papers and reports read before the Academy with the discussion of the same, and from them, with the aid of the Secretary (and with the sanction of the Council), compile and publish the Transactions.

The Board of Councilors

The Board of Councilors shall be the Board of Trustees of the Academy, and shall meet at the call of the President.

It shall be the Nominating Committee for Officers.

It shall be the Membership Committee and pass upon all applicants for Fellowship.

It shall consider all matters pertaining to the welfare of the Academy. It shall investigate all charges against members, and arbitrate and decide by a majority vote all professional controversies that may come before the Council, and its decisions on such questions shall be final. It shall appoint a successor or substitute to fill the place of an elective officer, who, for any reason, is unable, during the current year, to perform his duties.

The American Board for Ophthalmic Examinations

Three of the nine members of the American Board for Ophthalmic Examinations represent the Academy. According to the By-Laws of the Board, one member is elected each year to serve three years, beginning the first of January next following the meeting at which he is elected. No member of the Board may serve more than six years continuously. Members shall be chosen in the same manner as the presiding Officers of the Society which they represent.

In accordance with this arrangement, one Fellow shall be elected each year, on nomination by the Council, to serve for three years as a member of the American Board for Ophthalmic Examinations.

National Board of Examiners in Oto-Laryngology

A Committee of two Active Fellows of the Academy shall be appointed annually by the President, with the advice of the Council, to serve on the National Board of Examiners in Oto-Laryngology.

The Section on Instruction

The Section on Instruction shall be directed by four Active Fellows of the Academy, one of whom shall be elected each year, to serve for a period of four years.

The Program Committee

The Program Committee shall consist of the President, First Vice-President and Secretary, and it shall prepare the scientific program of the Annual Meetings.

The Committee on Exhibits

The Committee on Exhibits shall consist of one or more members and shall arrange an exhibit of instruments, pathologic and histologic specimens illustrating subjects connected with the eye, ear, nose and throat.

The Committee on Arrangements

The Committee on Arrangements shall provide for such social and other features as it may deem expedient.

The Necrologist

The Necrologist shall be appointed by the President, and he shall present at the annual meeting a report of the recent death of any Fellow and prepare a brief biographic sketch of same.

The Auditing Committee

The Auditing Committee shall consist of three Fellows appointed by the President, who shall audit the books of the Treasurer and present their report in writing to the Board of Councilors.

The Committees

The following Standing Committees shall be appointed by the President: The Committee on Arrangements, the Committee on Exhibits, the Examining Committee in Oto-Laryngology, and the Committee on Ophthalmic and Oto-Laryngic Pathology.

Article 5

Dues

The fiscal year of the Academy shall commence January first, and the current annual dues of ten dollars ($10.00) are payable at this time. The entrance fee paid by the newly elected Fellow shall cover his annual dues to December thirty-first following his election.

A copy of the Transactions of the preceding meeting will be forwarded free of charge to all Fellows whose annual dues are paid before March first.

A member in arrears for two years' dues, who fails after notification by Treasurer or Secretary to last address to pay such arrears, must be dropped from the roll.

Article 6

Reelection and Reinstatement

A Fellow whose name has been dropped from the roll for nonpayment of dues, as provided in Article 5, may be readmitted into Fellowship by formally applying for admission as a new member.

A Fellow who resigns from Fellowship in the Academy may be reinstated, at the discretion of the Council, by conforming to the requirements of the Society, as provided in Article 3 of the Constitution.

ARTICLE 7

ORDER OF BUSINESS

1. Meeting called to order.
2. Announcements.
3. Reading of Minutes.
4. Report of Secretary.
5. Report of Treasurer.
6. Report of Editor of the Transactions.
7. Report of Standing Committees:
   Program.
   Section on Instruction.
   Section on Ophthalmic and Oto-Laryngic Pathology.
   American Board for Ophthalmic Examinations.
   National Board of Examiners in Oto-Laryngology.
   Necrologist.
   Arrangements.
8. Address of President.
9. Reading of Papers.
10. Unfinished Business.
11. New Business.
12. Report of Council.
13. Election of Officers.

ARTICLE 8

AMENDMENTS

The By-Laws may be amended at any annual meeting by a two-thirds vote of the Fellows present, notice of the proposed amendment having been given at the previous annual meeting.

Roberts' Rules of Order shall be the parliamentary guide in the meetings of the Academy.

ALPHABETIC LIST

Honorary Fellows

DUNDAS-GRANT, SIR JAMES,
148 Harley St., W. 1, London, Eng.
ELLIOTT, LT.-COL. R. H.,
54 Welbeck St., Cavendish Sq., London, W., Eng.
GRAY, ALBERT A. . . . . Glasgow, Scotland
JOHNSON, LINDSAY. . . . Britannia Bldg., West St., Durban, Natal
ELSCHNIG, PROF. A.
Ferdinandstrasse, 10, Prague, Czecho-Slovakia
LAWFORD, J. B.,
99 Harley St., Cavendish Sq., W. London, Eng.
LISTER, SIR WILLIAM T.,
24 Devonshire Place, W. 1, London, Eng.
VAN DER HOEVE, PROF. J. . . Rijnsburgerweg, 6 A, Leiden, Holland

---

Life Members

BROWN, EDWARD J. . 900 Nicollet Ave., Minneapolis, Minn., OALR
*DAYTON, W. L. . . . . Funke Bldg., Lincoln, Neb., OALR
*FOSTER, HAL. . . . . 1014 Chambers Bldg., Kansas City, Mo., ALR
HOWE, LUCIEN. . . . . 520 Delaware Ave., Buffalo, N. Y., Op.
*MAYER, EMIL. . . . . 40 E. 41st St., New York City, N. Y., ALR
STANDISH, MYLES,
51 Hereford St., Back Bay, Boston, Mass., Op.
STEWART, T. M. . . 901 Union Trust Bldg., Cincinnati, O., OALR

Fellows

*ABBOTT, W. J. . . . . 8314 Euclid Ave., Cleveland, O., OALR
ADAMS, A. L. . . . . 323 West State St., Jacksonville, Ill., OALR
ADAMS, C. . . . . 52 West State St., Trenton, N. J., OALR
ADAMS, DANIEL S.,
520 Hume-Mansur Bldg., Indianapolis, Ind., ALR
ADAMS, ELDRIDGE S. . . . Moore Bldg., San Antonio, Tex., OALR
*ADAMS, FRANK M. . . . . 224 Thayer St., Providence, R. I., ALR
ADAMS, FRED'K W. . . . . Roosevelt Clinic, Seattle, Wash., OALR
AGNEW, FRED'K W. 120 3rd Ave., S. E., Independence, Ia., OALR
AINSLEE, GEORGE. . . 606 Oregonian Bldg., Portland, Ore., OALR
ALBRIGHT, LT. P. M., M. C., U. S. Navy, U. S. Naval Hosp.,
Brooklyn, N. Y., ALR

---

*Attended 1924 meeting.

ALDEN, ARTHUR M.,
3858 Westminster Place, St. Louis, Mo., ALR
ALDERDYCE, W. W.....Nasby Blk., Toledo, O., OALR
ALEXANDER, GEORGE JAMES,
1831 Chestnut St., Philadelphia, Pa., ALR
ALKIRE, H. L.....614 Kansas Ave., Topeka, Kan., OALR
ALLEN, CHAS. E..712 Waldheim Bldg., Kansas City, Mo., OALR
ALLEN, G. H.....Mills Bldg., Topeka, Kan., OALR
ALLEN, GEORGE V.....835 Kansas Ave., Topeka., Kan., OALR
ALLEN, J. H.....Majestic Bldg., Denver, Colo., ALR
ALLEN, L. P.....19 Jefferson Ave., Oshkosh, Wis., OALR
ALLEN, S. E.....11½ E. 8th St., Cincinnati, O., ALR
ALLPORT, F.....7 West Madison St., Chicago, Ill., OALR
ALTER, F. W.....Toledo Medical Bldg., Toledo, O., OALR
ALTRINGER, ARTHUR N.,
1100 Rialto Bldg., Kansas City, Mo., ALR
*ALWAY, ROBERT D...423 S. Lincoln St., Aberdeen, S. D., OALR
AMBERG, EMIL.....David Whitney Bldg., Detroit, Mich., ALR
ANDERSON, W. B.....507 Main Ave., Brownwood, Tex., OALR
ANDREWS, A. H.....30 N. Michigan Ave., Chicago, Ill., OALR
ANDREWS, B. F.....25 E. Washington St., Chicago, Ill., OALR
*ANDREWS, H. D.....23 Allen St., Buffalo, N. Y., Op.
ANDREWS, M. P.....Dempsey Bldg., Manitowoc, Wis., OALR
ANNON, WALTER T.....259 S. 17th St., Philadelphia, Pa., ALR
APPLEMAN, L. F..308 South 16th St., Philadelphia, Pa., Op.
*ARBUCKLE, M. F...University Club Bldg., St. Louis, Mo., ALR
*ARNOLD, F. J.....182 Pearl St., Burlington, Vt., OALR
*ARRELL, WILLIAM.....317 Main, E., Hamilton, Ontario, OALR
ARROWSMITH, H.....170 Clinton St., Brooklyn, N. Y., ALR
ASH, GARRETT G.....1 Main St., Bradford, Pa., OALR
ASHLEY, T. W.....466 Park Ave., Kenosha, Wis., OALR
ATKINSON, D. T..1515 Russell Bldg., San Antonio, Tex., OALR
AUERBACH, J...120 W. 86th St., New York City, N. Y., ALR
AUFMWASSER, H.....1302 Greenup St., Covington, Ky., OALR
AUTEN, F. E...1st National Bank Bldg., Belleville, Ill., OALR
AVERY, F. T.....357 W. 63rd St., Chicago, Ill., Op.LR
AYNESWORTH, H. T.....Amicable Bldg., Waco, Tex., OALR

*BABBITT, JAMES A..2117 Chestnut St., Philadelphia, Pa., ALR
BAHN, CHAS. A.,
722 Maison Blanche Annex, New Orleans, La., Op.
BAILEY, F. W..... Security Bldg., Cedar Rapids, Ia., OALR
BAILEY, H.....840 Landers Bldg., Springfield, Mo., OALR
BAIRD, CHAS. G.....Beatrice, Neb., OALR
BAKER, C. H.....Crapo Blk., Bay City, Mich., OALR
*BAKER, H. B.....59 Main St., Taunton, Mass., OALR
BALDWIN, KATE W....1117 Spruce St., Philadelphia, Pa., ALR
BALENTINE, P. L...1524 Chestnut St., Philadelphia, Pa., Op.
*BALL, J. MOORES.....4500 Olive St., St. Louis, Mo., Op.
BALL, M. V.....214 Penn Ave., W., Warren, Pa., OALR

*Ballenger, H. C.....25 E. Washington St., Chicago, Ill., ALR
Ballin, M. J.....108 E. 60th St., New York City, N. Y., ALR
Ballon, David H.....255 Bishop St., Montreal, Canada, ALR
Bane, Wm.....330 Metropolitan Bldg., Denver, Colo., OALR
Bane, W. C.....330 Metropolitan Bldg., Denver, Colo., OALR
*Banister, J. M.400 Brandeis Theater Bldg., Omaha, Neb., OALR
*Barber, F.....75 S. Fitzhugh St., Rochester, N. Y., Op.
Barck, C.....Humboldt Bldg., St. Louis, Mo., Op.A
Bardenheier, F. G. A.....Frisco Bldg., St. Louis, Mo., ALR
Barkan, A.....Credit Swisse Zürich, Switzerland, OALR
Barkan, H.....Physicians Bldg., San Francisco, Cal., Op.
Barker, Chas. B.,
224½ W. Oklahoma Ave., Guthrie, Okla., OALR
Barker, O. G. A...Johnstown Trust Co., Johnstown, Pa., Op.
Barnes, L. E.....6860 South Halsted St., Chicago, Ill., OALR
*Barnhill, J. F.....Pennway Bldg., Indianapolis, Ind., ALR
Barlow, Roy A.,
461 Farmers Trust Bldg., South Bend, Ind., ALR
Baron, F. S.....Peoples Bank Bldg., Zanesville, O., OALR
Bartholomew, A. C.,
122 E. Washington St., Ft. Wayne, Ind., OALR
Bass, A. L.....Francis Bldg., Louisville, Ky., OALR
Baum, H. L.....Metropolitan Bldg., Denver, Colo., ALR
*Beach, S. J.....704 Congress St., Portland, Me., OALR
Beane, George W..Jenkins Arcade Bldg., Pittsburgh, Pa., OALR
*Beattie, W. Henry.....252 Genesee St., Utica, N. Y., OALR
*Beattie, Robert...David Whitney Bldg., Detroit, Mich., OALR
Beatty, H. G.....150 E. Broad St., Columbus, O., OALR
Beaudoux, H. A.
401 La Salle Bldg., Minneapolis, Minn., Op.A
Beck, F. L.....408 Hynds Bldg., Cheyenne, Wyo., OALR
*Beck, J. C.....2551 N. Clark St., Chicago, Ill., ALR
Becker, H. M.....49 S. 4th St., Sunbury, Pa., OALR
*Bedell, A. J.....344 State St., Albany, N. Y., Op.A
*Beebe, C. S.....Wells Bldg., Milwaukee, Wis., OALR
Beggs, W. F.....2 Lombardy St., Albany, N. Y., OALR
Beil, J. Wallace...316 Argyle Bldg., Kansas City, Mo., OALR
Bell, Geo. Huston,
40 E. 41st St., New York City, N. Y., Op.
Bell, M. H...First Natl. Bank Bldg., Vicksburg, Miss., OALR
Bellin, Joseph.....1026 W. 6th St., Los Angeles, Cal., OALR
Bellows, George E.....Rialto Bldg., Kansas City, Mo., Op.
*Beem, I. F.....31 N. State St., Chicago, Ill., OALR
*Benedict, W. L.....Mayo Clinic, Rochester, Minn., Op.
Bennetto, Frederick R.,
172 Woolwich St., Guelph, Ont., Canada, OALR
Bennett, A. G.....26 Allen St., Buffalo, N. Y., Op.
*Bentley, Neil,
1161 David Whitney Bldg., Detroit, Mich., OALR

*Berens, Conrad, Jr.,
9 E. 46th St., New York City, N. Y., Op.
*Bergeron, J. Z.....104 S. Michigan Blvd., Chicago, Ill., ALR
*Bernatz, C. F.....Park Bldg., Pittsburgh, Pa., Op.
Bernstein, E. J.....Professional Bldg., Detroit, Mich., OALR
Berrisford, Paul D...812 Lowry Bldg., St. Paul, Minn., OALR
Berry, D. F.....Hume-Mansur Bldg., Indianapolis, Ind., ALR
Bettison, D. L.....502 Wilson Bldg., Dallas, Tex., OALR
Beyer, Louis J.....449 Delaware Ave., Buffalo, N. Y., ALR
Binger, H. E.....1027 Lowry Bldg., St. Paul, Minn., OALR
*Binkley, R. S...550 Fidelity Medical Bldg., Dayton, O., OALR
*Birkett, H. S.....252 Mountain St., Montreal, Canada, ALR
Bishop, C. W...616 La Salle Bldg., Minneapolis, Minn., OALR
Blaauw, E. E.....190 Ashland Ave., Buffalo, N. Y., OALR
Black, M.....Metropolitan Bldg., Denver, Colo., Op.
Black, N. M.....Wells Bldg., Milwaukee, Wis., Op.
Black, W. D.....Metropolitan Bldg., St. Louis, Mo., OALR
Blackburn, W. J.....661 Reibold Bldg., Dayton, O., OALR
*Blair, W. W.....Diamond Bank Bldg., Pittsburgh, Pa., Op.
Blake, W. F.....516 Sutter St., San Francisco, Cal., Op.
Blakesley, T. S.....Lathrop Bldg., Kansas City, Mo., OALR
*Blassingame, Charles D.,
Exchange Bldg., Memphis, Tenn., ALR
Bledsoe, R. W.....1005 Madison Ave., Covington, Ky., OALR
Blickenstaff, A. J...804 Peoria Life Bldg., Peoria, Ill., OALR
Bliss, C. B.....411 Columbus Ave., Sandusky, O., OALR
Blue, J. B.....Exchange Bldg., Memphis, Tenn., OALR
Blum, H. N...Maison Blanche Bldg., New Orleans, La., Op.
Bobb, E. V.....1st Natl. Bank Bldg., Mitchell, S. D., OALR
Boeckmann, Egil.....Lowry Bldg., St. Paul, Minn., OALR
Boehringer, H. W..1811 S. 22nd St., Philadelphia, Pa., Op.
*Boerner, Morris H....Scarborough Bldg., Austin, Tex., OALR
*Boiler, W. F.....426 S. Dodge St., Iowa City, Ia., OALR
Bone, H. D.....29 Parker St., Gardner, Mass., OALR
Boone, J. C...411 Citizens Bank Bldg., South Bend, Ind., OALR
Boone, James L.,
N. Y. Eye and Ear Inf., New York City, N. Y., ALR
Boot, G. W.....600 Main St., Evanston, Ill., OALR
*Bordley, James, Jr..330 N. Charles St., Baltimore, Md., Op.
Boucher, R. B.....414 Banks Bldg., Vancouver, B. C., OALR
Bourbon, Oliver P.,
222 Westlake Professional Bldg., Los Angeles, Cal., OALR
*Bousquet, J. P. E..26 Sherbrooke St., W., Montreal, Can., OALR
Bouvy, Harry.....New Foley Bldg., La Grange, Ore., OALR
Boyce, G. H.....408 E. D. St., Iron Mountain, Mich., OALR
Boyce, Wm. B.....1019 Ludington St., Escanaba, Mich., OALR
Boyd, E. T.....823 Majestic Bldg., Denver, Colo., OALR
Boyd-Snee, Harry,
716 L. M. S. Bldg., South Bend, Ind., ALR
Bozer, Herrmann E.,
438 Delaware Ave., Buffalo, N. Y., ALR

BRADFIELD, J. A. L....State Bank Bldg., La Crosse, Wis., OALR
BRASWELL, C. J.....726 Mayo Bldg., Tulsa, Okla., OALR
BRAUNLIN, WM. H.,
Marion Natl. Bank Bldg., Marion, Ind., OALR
*BRAWLEY, F. E.....30 N. Michigan Blvd., Chicago, Ill., OALR
BRAWLEY, R. V.....Wallace Bldg., Salisbury, N. C., OALR
BRAY, E. R.....1440 Lincoln Ave., St. Paul, Minn., OALR
BRECKENRIDGE, HARRY E....209 Sixth St., Racine, Wis., OALR
BREHMER, HARRISON L.,
521 Luna Blvd., Albuquerque, N. M., OALR
BREWER, JOHN D.....618 Main St., Dyersburg, Tenn., OALR
BRIBACH, E. J.....212 Simpson Bldg., Atchison, Kan., OALR
*BRICKLEY, D. W.....196 W. Center St., Marion, O., OALR
BRIGGS, FRANCIS W...400 Lyceum Bldg., Duluth, Minn., ALR
BRIGGS, H. H.....73 Haywood St., Asheville, N. C., OALR
BRIGGS, W. E.....Box 166, Sacramento, Cal., OALR
BRITTON, G. S.....University Blk., Syracuse, N. Y., OALR
BROBST, C. H.....Central Bank Bldg., Peoria, Ill., OALR
BRODRICK, F. W.....511 Lawrence Bldg., Sterling, Ill., OALR
BROOKS, E. B.....1010 Terminal Bldg., Lincoln, Neb., OALR
BROOKS, E. H.....808 College Ave., Appleton, Wis., OALR
BROSE, L. D.....501 Upper First St., Evansville, Ind., OALR
BROWN, C. M.....510 Delaware Ave., Buffalo, N. Y., ALR
BROWN, E. T.....30 Church St., Burlington, Vt., OALR
*BROWN, G. M.....802 University Bldg., Syracuse, N. Y., ALR
BROWN, H. A.....719 St. Paul Bldg., San Francisco, Cal., Op.
*BROWN, J. E.....370 East Town St., Columbus, O., OALR
BROWN, JOHN C....632 Lowry Bldg., St. Paul, Minn., OALR
BROWN, J. M.....1136 W. 6th St., Los Angeles, Cal., ALR
*BROWN, L. E.....2nd Natl. Bank Bldg., Akron, O., OALR
BROWN, MORROW D.,
806 Metropolitan Bldg., Denver, Colo., ALR
BROWN, SAMUEL HORTON,
1901 Mt. Vernon St., Philadelphia, Pa., Op.
*BROWN, W. E..8th Ave. and Amity St., Homestead, Pa., OALR
*BROWN, W. H.,
Bk. of Montreal Bldg., Edmonton, Alberta, Can., OALR
BRUDER, J.....229 W. 79th St., New York City, N. Y., OALR
BRUÉRE, G. E.....Journal Bldg., Portland, Ore., OALR
BRUMM, SETH A.,
818 Stock Exchange Bldg., Philadelphia, Pa., ALR
BRUNER, A. B.....Guardian Bldg., Cleveland, O., Op.
BRUNER, W. E.....Guardian Bldg., Cleveland, O., Op.
BRUST, H. O.....720 S. Crouse Ave., Syracuse, N. Y., ALR
BRYAN, W. M. C.,
University Club Bldg., St. Louis, Mo., ALR
*BRYANT, A. G.....502 Beacon St., Boston, Mass., ALR
BUCHANAN, JAMES N.....Tarbox Bldg., Freeport, Ill., OALR
BUCK, R. H.....Venetian Bldg., Chicago, Ill., OALR
BUCKLEY, J. H.....1st Natl. Bk. Bldg., Ft. Smith, Ark., OALR

BUCKMAN, E. V...70 S. Franklin St., Wilkes-Barre, Pa., OALR
BULL, E. L.....124 E. State St., Ithaca, N. Y., OALR
*BULSON, A. E., JR....406 W. Berry St., Ft. Wayne, Ind., OALR
BURCH, F. E.....Hamm Bldg., St. Paul, Minn., A
BURKE, T. A.....446 Rose Bldg., Cleveland, O., OALR
BURLESON, J. H..Central Trust Bldg., San Antonio, Tex., OALR
BURNS, S. S.....Humboldt Bldg., St. Louis, Mo., ALR
BURROUGHS, G. M.....5 Broad St., Danielson, Conn., Op.
BURTON, F. A.....404 Watts Bldg., San Diego, Cal., ALR
*BUTLER, MARGARET F.,
1831 Chestnut St., Philadelphia, Pa., ALR
BUTLER, ROBERT H.,
227 W. Columbus Ave., Bellefontaine, O., OALR
BUTT, M. M.....2045 Chestnut St., Philadelphia, Pa., Op.
BUVINGER, CHARLES W.,
50 Washington St., East Orange, N. J., OALR
*BYRNES, H. F.....4 Chestnut St., Springfield, Mass., OALR

*CAHILL, J. W.....390 Main St., Worcester, Mass., OALR
CALDWELL, R...State Natl. Bank Bldg., Little Rock, Ark., OALR
CALHOUN, F. P.....Candler Bldg., Atlanta, Ga., OALR
CALLFAS, W. F.....567 Brandeis Bldg., Omaha, Neb., ALR
CAMERON, WILLIAM G.,
1404 National Realty Bldg., Tacoma, Wash., OALR
CAMP, W. E...910 Donaldson Bldg., Minneapolis, Minn., OALR
*CAMPBELL, C. H.,
Columbus State Bk. Bldg., Columbus, Neb., OALR
*CAMPBELL, D. A...David Whitney Bldg., Detroit, Mich., OALR
CAMPBELL, D. M.....Peter Smith Bldg., Detroit, Mich., OALR
CAMPBELL, ROBERT A.,
520 LaSalle Bldg., Minneapolis, Minn., OALR
*CAMPBELL, WM. KING.96 Third Ave., Long Branch, N. J., OALR
CAPRON, E. P.....118 Angell St., Providence, R. I., OALR
CARLETON, RALPH.....10 Temple St., Springfield, Mass., Op.
CARMACK, J. W...37 Willoughby St., Indianapolis, Ind., AR
*CARMODY, T. E.....Metropolitan Bldg., Denver, Colo.,ALR
CARNCROSS, HELEN..22 E. Washington St., Chicago, Ill., Op.
CARPENTER, E. R..708 Medical Arts Bldg., Dallas, Tex., OALR
CARPENTER, E. W....201 E. North St., Greenville, S. C., ALR
CARR, G. W.....54 S. Franklin St., Wilkes-Barre, Pa., OALR
CARROLL, F.....Ely Blk., Cedar Rapids, Ia., OALR
*CARROLL, G. G.....614 Main St., West, Rochester, N. Y., OALR
CARSON, W. E....Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
*CARTER, W. W...24 E. 48th St., New York City, N. Y., ALR
*CARVILL, L. MAUD,
82 Commonwealth Ave., Boston, Mass., Op.
*CARY, E. H.....Southwestern Life Bldg., Dallas, Tex., OALR
CARY, G. C.....Grand Junction, Colo., OALR
*CAVANAUGH, J. A.....7 W. Madison St., Chicago, Ill., ALR
CAYCE, E. B.....Hitchcock Bldg., Nashville, Tenn., OALR

CHADWICK, IRA B...4 Terminal Bldg., Coffeyville, Kan., OALR
CHAMBERS, T. R.....15 Exchange Pl., Jersey City, N. J., OALR
*CHAPMAN, V. A....Colby-Abbot Bldg., Milwaukee, Wis., OALR
*CHARLES, J. W.....Humboldt Bldg., St. Louis, Mo., Op.
*CHARLTON, C. C..124 S. Illinois St., Atlantic City, N. J., ALR
CHASE, E. FRANK.....1008 Cobb Bldg., Seattle, Wash., OALR
CHASE, WALTER D....230 E. Broad St., Bethlehem, Pa., ALR
CHATTIN, J. FRANKLIN...671 Broad St., Newark, N. J., Op.
CHENERY, W. E..377 Commonwealth Ave., Boston, Mass., ALR
CHENEY, F. E...64 Commonwealth Ave., Boston, Mass., Op.
CHENEY, JAMES W...702 Schweiter Bldg., Wichita, Kan., OALR
*CHILDS, H. A.....Eagle Blk., Creston, Ia., OALR
*CLARK, F. T.....92 Broad St., Westfield, Mass., OALR
*CLARK, H. S.....Syndicate Blk., Minneapolis, Minn., OALR
CLARK, I. G.....188 E. State St., Columbus, O., OALR
CLARK, J. S.....67 Stephenson St., Freeport, Ill., OALR
*CLARK, L. H.....37 Monroe Ave., Rochester, N. Y., OALR
CLAY, G. E.....426 Candler Bldg., Atlanta, Ga., OALR
CLERF, LOUIS H.....128 S. 10th St., Philadelphia, Pa., ALR
CLEVENGER, W. F.,
Newton Claypool Bldg., Indianapolis, Ind., ALR
COATES, G. M.....1736 Pine St., Philadelphia, Pa., ALR
COBB, EDWIN.....Masonic Temple, Marshalltown, Ia., OALR
COCHRAN, R. W.....West and 3rd Sts., Madison, Ind., OALR
COFFELT, THEODORE A..Woodruff Bldg, Springfield, Mo., OALR
*COGAN, J. E.....Rose Bldg., Cleveland, O., OALR
*COHEN, MARTIN,
1 West 85th St., New York City, N. Y., OALR
COHEN, SAMUEL.....1626 Spruce St., Philadelphia, Pa., ALR
COHN, F.....31 East 60th St., New York City, N. Y., ALR
COHN, S.....416 Luke St., Fulton, Ky., OALR
COLLINS, F. W.....Majestic Bldg., Denver, Colo., ALR
COLLINS, H.....New Jersey Bldg., Duluth, Minn., OALR
COLLINS, J. D.....90 Main St., Northampton, Mass., ALR
*COLVER, B. N..182 Manchester St., Battle Creek, Mich., OALR
COMEE, W. C.....Bellin Bldg., Green Bay, Wis., OALR
CONLON, F. A.....Bay State Bldg., Lawrence, Mass., OALR
CONNOR, CHAS. E.....Hamm Bldg., St. Paul, Minn., OALR
*CONNOR, RAY.....1410 Stroh Bldg., Detroit, Mich., OALR
COOK, S. E.....Richards Blk., Lincoln, Neb., OALR
COOK, W. ALBERT.....506 Palace Bldg., Tulsa, Okla., OALR
COOPER, C. E.....Metropolitan Bldg., Denver, Colo., ALR
*COOVER, D. H.....Metropolitan Bldg., Denver, Colo., Op.
COPPS, LYMAN A.....Marshfield, Wis., OALR
*CORSER, J. B..Scranton Private Hospital, Scranton, Pa., OALR
COTT, C. C.....483 Delaware Ave., Buffalo, N. Y., ALR
*COTT, DELZON N.....1001 Main St., Buffalo, N. Y., ALR
COULTER, C. F...1st Natl. Bk. Bldg., Great Falls, Mont., OALR
COVINGTON, L. C.,
217½ Capitol Ave., Charleston, W. Va., OALR

*Cowper, H. W.....543 Franklin St., Buffalo, N. Y., Op.
Craighead, Nancy B.,
7002 Jenkins Arcade Bldg., Pittsburgh, Pa., ALR
Craft, Kenneth L.,
226 Hume-Mansur Bldg., Indianapolis, Ind., OALR
Crampton, G. S.....1700 Walnut St., Philadelphia, Pa., Op.
Crane, J. Wellington...128 Third St., Trenton, N. J., ALR
Creighton, W. J..1905 Chestnut St., Philadelphia, Pa., Op.
Crisp, W. H.....Metropolitan Bldg., Denver, Colo., Op.
*Crockett, R. L.....Oneida, N. Y., Op.
Croman, Joseph M. Jr.,
Washington Apts., Mount Clemens, Mich., OALR
Croskey, John Welch,
1909 Chestnut St., Philadelphia, Pa., OALR
*Cross, A. E.....Slater Bldg., Worcester, Mass., Op.
Cross, G. H.....525 Welsh St., Chester, Pa., Op.
Crossley, E. R.....29 E. Madison St., Chicago, Ill., OALR
Crump, J. F..507 Citizens Bank Bldg., Pine Bluff, Ark., OALR
Culbertson, L. R.....Masonic Temple, Zanesville, O., OALR
Cullom, Marvin, McT.,
208 Hitchcock Bldg., Nashville, Tenn., OALR
Cushman, Beulah...25 E. Washington St., Chicago, Ill., Op.
Curdy, R. J.....1134 Rialto Bldg., Kansas City, Mo., Op.
Curran, Edw. J..401 Waldheim Bldg., Kansas City, Mo., Op.
Curry, G. E.....Westinghouse Bldg., Pittsburgh, Pa., Op.
Curtin, Eugene A...703 Connell Bldg., Scranton, Pa., OALR
Curtis, W. L.,
612 Security Mutual Bk. Bldg., Lincoln, Neb., OALR
*Cunningham, Allan R.,
260 Barrington St., Halifax, N. S., OALR
Cutler, Franklin E..1025 Schofield Bldg., Cleveland, O., ALR

*Dabney, V.....1633 Connecticut St., Washington, D. C., ALR
Dabney, S. G.....Starks Bldg., Louisville, Ky., OALR
Dailey, Gilbert L.....713 N. 3rd St., Harrisburg, Pa., OALR
Dancy, A. B.....First Natl. Bank Bldg., Jackson, Tenn., OALR
Daniels, R. L.....Elks Temple, New Bern, N. C., OALR
Darmer, George A....401 Terminal Bldg., Aurora, Ill., OALR
Davidson, Herman P..22 E. Washington St., Chicago, Ill., Op.
*Davies, T. Alex.....578 Sherbourne St., Toronto, Can., OALR
*Davis, A. E.....47 E. 57th St., New York City, N. Y., Op.
Davis, Frank C.....Lewistown, Mont., ALR
Davis, G. E.....42 W. 77th St., New York City, N. Y., OALR
Davis, Harry B...611 Lathrop Bldg., Kansas City, Mo., OALR
Davis, J. E.....Kress Bldg., McAlister, Okla., OALR
Davis, J. L.....135 S. 18th St., Philadelphia, Pa., ALR
*Davis, Ralph E.....1104 Selling Bldg., Portland, Ore., OALR
Davis, Thomas W.,
O'Hanlon Bldg., Winston-Salem, N. C., OALR
*Davis, W. Thornwall,
927 Farragut Sq., Washington, D. C., Op.

DAY, EWING W....Westinghouse Bldg., Pittsburgh, Pa., ALR
DAYTON, G. O.....Lewisohn Bldg., Butte, Mont., OALR
*DEAN, ALFRED..Gilbert Blk. Bldg., Grand Rapids, Mich., Op.
*DEAN, F. W.....Natl. Bank Bldg., Council Bluffs, Ia., OALR
DEAN, L. W.....12 S. Clinton St., Iowa City, Ia., OALR
DEAN, WALTER.....Francis Bldg., Louisville, Ky., OALR
DEBOE, M. P.....621 Eaton St., Key West, Fla., OALR
DECHERD, H. B.....Wilson Bldg., Dallas, Tex., OALR
DECKER, J. C.....Francis Bldg., Sioux City, Ia., OALR
*DEFNET, W. A.....62 Adams St., W., Detroit, Mich., ALR
DEICHLER, LYN WALLER,
2028 Chestnut St., Philadelphia, Pa., OALR
DENMAN, IRA O.....425 Ohio Bldg., Toledo, O., OALR
*DENNIS, D. N.....221 W. Ninth St., Erie, Pa., Op.A
DENNIS, E. P.....311 W. 10th St., Erie, Pa., OALR
*DENNIS, F. L...Burns Bldg., Colorado Springs, Colo., ALR
DENNIS, G. J.....25 E. Washington St., Chicago, Ill., ALR
DERBY, G. S.....23 Bay State Rd., Boston, Mass.,Op.
DETLING, F.....Investment Bldg., Los Angeles, Cal., OALR
DEWEESE, CLARENCE..154 N. Upper St., Lexington, Ky., OALR
DEWEY, C. H.,
Walter Reed General Hospital, Washington, D. C.
DICKINSON, B. M.,
5634 Stanton Ave., E. Liberty Sta., Pittsburgh, Pa., ALR
DINTENFASS, HENRY.....1714 Pine St., Philadelphia, Pa., ALR
DIXON, G. S.....40 E. 41st St., New York City, N. Y., R
DIXON, W. E.,
706 1st Natl. Bank Bldg., Oklahoma City, Okla., OALR
DOANE, L. I.....Reiber Bldg., Butler, Pa., OALR
DODD, O.....708 Church St., Evanston, Ill., Op.
DONNELL, NEWMAN R.,
Metropolitan Bldg., St. Louis, Mo., Op.
DONOHER, W. D.....Boston Bldg., Salt Lake City, Utah, OALR
DONOVAN, J. A.....Phoenix Bldg., Butte, Mont., OALR
DOLMAN, PERCIVAL.....Flood Bldg., San Francisco, Cal., Op.
DORENTO, DRED R.,
Merchants Natl. Bank Bldg., Fort Smith, Ark., OALR
DORGE, RICHARD I.,
76 Seymour Ave., S. E., Minneapolis, Minn., OALR
DORSEY, J. G.....201 N. Main St., Wichita, Kan., OALR
DOUGLAS, F. A.....State Bk. Bldg., La Crosse, Wis., OALR
DOW, F. E.....4 West St., Northampton, Mass., Op.
*DOWLING, J. I.....116 Washington Ave., Albany, N. Y., OALR
DOWLING J. T.....Cobb Bldg., Seattle, Wash., OALR
*DUANE, A.....139 E. 37th St., New York City, N. Y., Op.
DUANE, J. F.....Jefferson Bldg., Peoria, Ill., OALR
DUCKWORTH, GUILFORD M.....Buchel Blog., Cuero, Tex., OALR
DUDLEY, W. H....Brockman Bldg., Los Angeles, Cal., OALR
DULANEY, O.....717 Starks Bldg., Louisville, Ky., OALR
DUNLAP, LAWRENCE G.,
Electric Light Bldg., Anaconda, Mont., OALR

DUNN, I. J.....Masonic Temple, Erie, Pa., OALR
DUNN, O. B.....4th and Railroad Sts., Ironton, O.
*DUNNINGTON, JOHN H.,
104 E. 40th St., New York City, N. Y., Op.
DUNTLEY, G. S.....220 E. Carroll St., Macomb, Ill., OALR
*DUTROW, H. V..1040 Fidelity Medical Bldg., Dayton, O., OALR
DUTTON, C. K.....Moberly, Mo., OALR
DWIGHT, C. G.....Pioneer Bldg., Madison, Wis., OALR
DWORETZKY, JULIUS.....7 Law St., Liberty, N. Y., ALR
DYER, CLYDE P.....Metropolitan Bldg., St. Louis, Mo., Op.

EAREL, A. M.....507 Wildon Blk., Hoopeston, Ill., OALR
EARL, HARRY D.....3717 Spruce St., Philadelphia, Pa., ALR
EASTON, J. C.....Fairbanks Bldg., Springfield, O., OALR
EDSON, R.....560 Delaware Ave., Buffalo, N. Y., Op.
EDWARDS, C. J.....1422 Wash St., Vicksburg, Miss., OALR
EDWARDS, S. R...111 N. Lafayette St., South Bend, Ind., OALR
EDWARDS, W. A.....329 Main St., La Crosse, Wis., OALR
ELLEGOOD, J. A.....Equitable Bldg., Wilmington, Del., OALR
*ELLETT, E. C.....Exchange Bldg., Memphis, Tenn., Op.
ELWOOD, CALVIN R.....Spies Bldg., Menominee, Mich., OALR
*EMERSON, L.....Metropolitan Bldg., Orange, N. J., OALR
ENGLE, H. P.....200 Olive St., Newton, Ia., OALR
ERSNER, MATTHEW S....1729 Pine St., Philadelphia, Pa., ALR
ESTABROOK, C. T.....390 Main St., Worcester, Mass., OALR
ESTERLY, D. E.....Mills Bldg., Topeka, Kan.
EVENSEN, H. O.....25 E. Washington St., Chicago, Ill., OALR
*EWING, ARTHUR E...Metropolitan Bldg., St. Louis, Mo., Op.

FAIRBAIRN, JOHN....503 Delaware Ave., Buffalo, N. Y., ALR
*FAIRCHILD, NORA M.,
600 Brandeis Theater Bldg., Omaha, Neb., Op.
*FAIRING, JOHN W...204 Tribune Bldg., Greensburg, Pa., OALR
*FAITH, THOMAS.....31 N. State St., Chicago, Ill., OALR
*FALLOWS, H. D...Park Hospital Clinic, Mason City, Ia., OALR
FARRELL, P. J.....25 E. Washington St., Chicago, Ill., OALR
*FARMER, A. G.....1040 Fidelity Med. Bldg., Dayton, O., OALR
FARNESWORTH, M. A.,
146 Greenwood St., Battle Creek, Mich., OALR
FARNSLER, H. H.....1438 Market St., Harrisburg, Pa., OALR
FARRINGTON, P. M.,
1473 Goodbar Ave., Memphis, Tenn., ALR
*FAWCETT, I.....Schmulbach Bldg., Wheeling, W. Va., OALR
*FEINGOLD, M..4206 St. Charles Ave., New Orleans, La., Op.
FELLOWS, G. G.....30 N. Michigan Blvd., Chicago, Ill., OALR
*FELT, CARL L.,
N. E. Cor. Chestnut & 20th Sts., Philadelphia, Pa., ALR
FENTON, RALPH A.....616 Journal Bldg., Portland, Ore., OALR
FERNANDEZ, FRANCISCO M.....Prado 105, Havana, Cuba, OALR
FERGUSON, E. I.,
607 1st Natl. Bank Bldg., Oklahoma City, Okla., OALR

FERMAN, JOHN W.....Stahlman Bldg., Charleroi, Pa., OALR
FINNOFF, W. C.....Imperial Bldg., Denver Colo., Op.
FISCHER, V. B.....Mercantile Bk. Bldg., Boulder, Colo., OALR
FISHER, CARL..1028 Pacific Mut. Bldg., Los Angeles, Cal., Op.A
*FISHER, WM. A.....31 N. State St., Chicago, Ill., OALR
FISKE, G. F.....25 E. Washington St., Chicago, Ill., OALR
FLAGG, J. D.....473 Virginia St., Buffalo, N. Y., Op.A
FLATLEY, T. J.....Reliance Bldg., Moline, Ill., OALR
FLOYD, T. W.....Lehman Bldg., Peoria, Ill., OALR
FLYNN, JAMES A..1616 Eye St., N. W., Washington, D. C., ALR
FOGARTY, CHAS. W..1826 Summit Ave., St. Paul, Minn., OALR
FOLEY, J. W.....Majestic Bldg., Denver, Colo., OALR
FORBES, H. H.....40 E. 41st St., New York City, N. Y., ALR
*FORBES, S. B.....409 Citizens Bk. Bldg., Tampa, Fla., OALR
*FORGRAVE, L. ROBERT.....Logan Bldg., St. Joseph, Mo., OALR
FORSYTHE, S. T.....1011 Euclid Ave., Cleveland, O., OALR
FORT, A. G.....833 Candler Bldg., Atlanta, Ga., OALR
FOSTER, J. H.....1300 Walker Ave., Houston, Tex., ALR
FOSTER, J. M.....Metropolitan Bldg., Denver, Colo., OALR
FOUTS, JOHN D.,
1110 Fidelity Medical Bldg., Dayton, O., OALR
FOWLER, S. R.....University Bldg., Syracuse, N. Y., ALR
FOX, CHAS. H.....2116 Central Ave., Kearney, Neb., OALR
FOX, L. W.....303 S. 17th St., Philadelphia, Pa., Op.
FOX, R. D.....411 Unity Bldg., Bloomington, Ill., OALR
FRANCHERE, FRED'K E.,
4th and Nebraska Sts., Sioux City, Ia., OALR
*FRANCIS, LEE M.....636 Delaware Ave., Buffalo, N. Y., Op.
FRANK, A. C..308 Diamond Bank Bldg., Pittsburgh, Pa., ALR
FRANK, IRA.....104 S. Michigan Blvd., Chicago, Ill., ALR
FREEDMAN, L. M.....419 Boylston St., Boston, Mass., ALR
FRELIGH, C. A.....75 State St., Springfield, Mass., ALR
*FRENCH, R. F,
Masonic Temple Bldg., Marshalltown, Ia., OALR
FREUDENTHAL, W.,
24 W. 88th St., New York City, N. Y., ALR
FREY, C. L.....529 Pine St., Scranton, Pa., OALR
FRIDENBERG, P. H..38 W. 59th St., New York City, N. Y., OALR
FRIEDENWALD, H.....1029 Madison Ave., Baltimore, Md., Op.A
FRIEDMAN, D.....44 W. 77th St., New York City, N. Y., ALR
FRINGER, W. R.....Wm. Brown Bldg., Rockford, Ill., Op.
FULLENWIDER, C. M.....Barnes Bldg., Muskogee, Okla., OALR
*FULLER, T. E.,
Texarkana Natl. Bank Bldg., Texarkana, Tex., OALR
FULKERSON, C. B.,
Kalamazoo Natl. Bank Bldg., Kalamazoo, Mich., OALR
FULTON, JOHN E.....728 Lowry Bldg., St. Paul, Minn., OALR
FURGASON, A. P.....Richards Blk., Lincoln, Neb., OALR
*FUST, JOHN H.....138 W. 9th St., Erie, Pa., OALR

GADBOIS, ARTHUR E....421 Norfolk Ave., Norfolk, Neb., OALR
GAILEY, WILLIAM W.,
615 Griesheim Bldg., Bloomington, Ill., OALR
GALE, M. JEAN.....Imperial Bldg., Denver, Colo., Op.
GALLAHER, T. J.....California Bldg., Denver, Colo., ALR
GARBER, J. M..... 48 Park Ave., Mansfield, O., OALR
GARRAGHAN, E. F...25 E. Washington St., Chicago, Ill., OALR
GARRETT, J. R.....Strickland Bldg., Roanoke, Va., OALR
GATEWOOD, W. L..15 E. 48th St., New York City, N. Y., ALR
GAVIN, E. F.....1 S. Sheridan Rd., Waukegan, Ill., OALR
GEIGER, C. W.....Cobb Bldg., Kankakee, Ill., OALR
GEORGE, H. W.....19 N. Union St., Middletown, Pa., Op.
GIBSON, R. D..Dollar Savings & Trust, Youngstown, O., OALR
GIFFORD, H.....Brandeis Bldg., Omaha, Neb., Op.A
GIFFORD, SANFORD R.,
702 Brandeis Theater Bldg., Omaha, Neb., Op.
GILBERT, F. Y.....148 Park St., Portland, Me., Op.AR
GILL, E. G.....McBain Bldg., Roanoke, Va., ALR
GILL, G.....146 Middle Ave., Elyria, O., OALR
*GILL, G. BACHE.....1624 Eye St., Washington, D. C., ALR
GILLETT, WILBUR G.....201 N. Main St., Wichita, Kas., Op.
GILLUM, JOHN R.....221 S. 6th St., Terre Haute, Ind., OALR
GITTENS, T. R.....Sioux City, Ia., OALR
GLASFORD, SAMUEL T.....Pekin, Ill., OALR
GLEASON, E. B.....2033 Chestnut St., Philadelphia, Pa., OALR
GLOGAU, O.....64 E. 91st St., New York City, N. Y., OALR
*GLOSSER, H. H.....448 Franklin St., Buffalo, N. Y., OALR
GLOVER, S. P.....1118 Twelfth Ave., Altoona, Pa., OALR
GODDARD, H. M.....1531 Spruce St., Philadelphia, Pa., ALR
GOLDBACH, L. J.....1012 N. Charles St., Baltimore, Md., OALR
*GOLDMAN, HARRY G...57 W. 58th St., New York, N. Y., ALR
*GOLDSMITH, P. G.....84 Carlton St., Toronto, Can., ALR
GOLDSTEIN, M. A..3858 Westminster Pl., St. Louis, Mo., ALR
GOODYEAR, HENRY M.....8 E. 8th St., Cincinnati, O., ALR
GORRELL, T. J.,
Vincennes and 16th Sts., Chicago Heights, Ill., OALR
*GORDAN, J. S.....309 Goldsmith Bldg., Milwaukee, Wis., OALR
GOSNEY, CHAS. W..6075 Franklin Ave., Los Angeles, Cal., OALR
GOTTLIEB, M. J.,
814 West End Ave., New York City, N. Y., ALR
GOURLEY, G. F.....407 Sinclair Bldg., Steubenville, O., OALR
*GOUX, R. S...545 David Whitney Bldg., Detroit, Mich., OALR
*GRACEY, GEORGE F.....209 State St., Harrisburg, Pa., OALR
*GRADLE, HARRY S.....110 N. Wabash Ave., Chicago, Ill., Op.
GRAHAM, H. B.....210 Post St., San Francisco, Cal., ALR
GRANGER, WAYNE B.705 Commercial St., Emporia, Kas., OALR
*GRANT, F. E..Kal. Natl. Bank Bldg., Kalamazoo, Mich., OALR
*GRANT, P. T.,
205 Widdicomb Bldg., Grand Rapids, Mich., OALR
GRAVES, W. H.....Commerce Bldg., Pittsburg, Kan., OALR
GREEN, A. S.....853 Hyde St., San Francisco, Cal., Op.

*Green, J., Jr.....Metropolitan Bldg., St. Louis, Mo., Op.
Green, L. D.....853 Hyde St., San Francisco, Cal., Op.
Greene, Jos. B.....Haywood Bldg., Asheville, N. C., ALR
*Greenwood, A..82 Commonwealth Ave., Boston, Mass., Op.
Greever, Boyd L.,
309 State Exchange Bldg., Hutchinson, Kan., OALR
Gregg, John B.,
Security Natl. Bank Bldg., Sioux Falls, S. D., OALR
Grim, U. J.....30 N. Michigan Blvd., Chicago, Ill., OALR
Griscom, J. Milton.1925 Chestnut St., Philadelphia, Pa., ALR
Gross, Julius E.,
8030 Jenkins Arcade Bldg., Pittsburgh, Pa., ALR
Grosvenor, L. N.....378 Dakota St., Huron, S. D., OALR
Grove, W. E.....1330 Wells Bldg., Milwaukee, Wis., OALR
Grushlaw, I.271 Central Pk., W., New York City, N. Y., ALR
Gsell, J. F.....911 Beacon Bldg., Wichita, Kas., OALR
Guerinot, Albert J.,
7094 Jenkins Arcade Bldg., Pittsburgh, Pa., OALR
Guggenheim, L. K....Carleton Bldg., St. Louis, Mo., ALR
Gundelach, C. A.,
825 University Club Bldg., St. Louis, Mo., ALR
Guthrie, A. L.,
Amer. Natl. Bk. Bldg., Oklahoma City, Okla., OALR
Guthrie, J. M....Rosenbaum Bldg., Meridian, Miss., OALR
Guttman, J..987 Madison Ave., New York City, N. Y., OALR
Guyton, B. S.....Carter Bldg., Oxford, Miss., OALR
Gwinn, G. E.....Hicks Bldg., San Antonio, Tex., OALR

Haas, Fred'k J....Masonic Bldg., Leavenworth, Kas., OALR
Hackney, F. J.,
630 Volunteer Bldg., Chattanooga, Tenn., OALR
Haden, H. C.913 Union Natl. Bk. Bldg., Houston, Tex., OALR
*Hager, W. A..111 N. Lafayette St., South Bend, Ind., OALR
Hagler, A. L.....Hagler Bldg., Springfield, Ill., OALR
Hagler, E. E.....Hagler Bldg., Springfield, Ill., OALR
Hall, Alice K....25 E. Washington St., Chicago, Ill., ALR
*Hall, Edw. P....124 Hunter Ave., Kansas City, Mo., OALR
*Hall, G. C.....Gaulbert Bldg., Louisville, Ky., OALR
Halsted, F. S..708 Metropolitan Bldg., Denver, Colo., OALR
Hampton, R. R...Boston Bldg., Salt Lake City, Utah, OALR
Hancock, J. C...135 Cambridge Pl., Brooklyn, N. Y., Op.
*Haney, Wm. P.,
703 Brandeis Theater Bldg., Omaha, Neb., OALR
Hans, C. L.....19 W. 7th st., Cincinnati, O., OALR
Hansen, Erling W.,
910 Donaldson Bldg., Minneapolis, Minn., OALR
*Harbert, J. P.....Canby Block, Bellefontaine, O., OALR
Hardesty, J. F.311 Metropolitan Bldg., St. Louis, Mo., Op.
Hardy, W. F.....Humboldt Bldg., St. Louis, Mo., Op.
Harkness, G. F.....2130 Main St., Davenport, Ia., OALR

HARRIS, C. M..Johnstown Trust Co., Johnstown, Pa., OALR
HARRIS, H. B.....Fidelity Med. Bldg., Dayton, O., OALR
HARRIS, W. C.....Groton Bldg., Cincinnati, O., ALR
HARRIS, W. ROBIN..Exchange Bldg., Memphis, Tenn., ALR
HARRISON, W. G.....Empire Bldg., Birmingham, Ala., OALR
HARTLEY, F. A.....Fairbanks Bldg., Springfield, O., OALR
HARTZ, H. J.....27 E. Adams St., Detroit, Mich., ALR
HARTZEL, SOL. M...Dollar Bank Bldg., Youngstown, O., OALR
HARVEY, N. D...114 Waterman St., Providence, R. I., OALR
HASKELL, A. D.....145 High St., Portland, Me., Op.
HASKIN, W. H.....40 E. 41st St., New York., N. Y., ALR
HASPEL, D. M.,
1214 Maison Blanche Bldg., New Orleans, La., OALR
HATCH, C. B.....3 W. Church St., Newark, O., OALR
HAUER, A. M.....327 E. State St., Columbus, O., OALR
HAUGHEY, W.....24 W. Main St., Battle Creek, Mich., OALR
HAWLEY, C. W...30 N. Michigan Blvd., Chicago, Ill., OALR
HAWMAN, ERLE G.,
4051 N. Broad St., Philadelphia, Pa., OALR
*HAYDEN, AUSTIN A.,
25 E. Washington St., Chicago, Ill., OALR
*HAYS, H. N.....22 W. 47th St., New York City, N. Y., ALR
HAZLETT, L. R.....117 S. Main St., Butler, Pa., OALR
HEALEY, JOHN F....503 Delaware Ave., Buffalo, N. Y., ALR
HEARD, C. F.....Masonic Temple Bldg., Erie, Pa., OALR
HEARD, MARY K.Commonwealth Bldg., Des Moines, Ia., Op.
HEATH, PARKER..David Whitney Bldg., Detroit, Mich., Op.
HECKEL, E. B..Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
HEEB, H. J.....Loan & Trust Bldg., Milwaukee, Wis., OALR
HEED, C. R.....1205 Spruce St., Philadelphia, Pa., Op.
HEGGIE, N. M.....Buckman Bldg., Jacksonville, Fla., OALR
HEITGER, J. D.....808 Francis Bldg., Louisville, Ky., OALR
*HEMPSTEAD, BERT E...Mayo Clinic, Rochester, Minn., ALR
*HENNESSEY, WM. W.....333 Essex St., Salem, Mass., OALR
HENNING, CARL.The Rochambeau, Washington, D. C., Op.
HENNINGER, L. L..205 Citizens Bk. Bldg., Pasadena, Cal., OALR
HENTON, GEO. E...412 Morgan Bldg., Portland, Ore., OALR
HERBERT, A. W.119 E. Lehigh Ave., Philadelphia, Pa., OALR
HERRIMAN, L. L.....Physicians Bldg., Alamosa, Colo., OALR
HESS, W. L.....1554 California St., Denver, Colo., OALR
*HETRICK, LLEWELLYN E.,
20 East 53d St., New York, N. Y., ALR
HETZEL, C. C.....Eccles Bldg., Ogden, Utah, OALR
HIGBEE, E. H...Grand and Olive Sts., St. Louis, Mo., Op.
HIGGINBOTHAM, T. L.,
Solomon Clinic Bldg., Wichita, Kans., OALR
*HIGGINS, REUBEN P.....20 Court St., Cortland, N. Y., OALR
*HIGGINS, S. G.....Wells Bldg., Milwaukee, Wis., OALR
HILL, A. J....301 Citizens Natl. Bk. Bldg., Salem, O., OALR
*HILL, CLARENCE E.....160 Bloor St., Toronto, Can., Op.
*HILL, EMORY.....Professional Bldg., Richmond, Va., Op.

*Hinman, Eugene E.,
146 Washington Ave., Albany, N. Y., ALR
Hinnen, G. A.....1343 Delta Ave., Cincinnati, O., OALR
Hitz, H. B.....Goldsmith Bldg., Milwaukee, Wis., ALR
Hobart, Bertha King,
304 Pro. Exch. Bldg., Toledo, O., OALR
Hodson, B. F.....25 N. E. First Ave., Miami, Fla., OALR
Hoffman, Walter F.,
916 Leary Bldg., Seattle, Wash., OALR
Hogan, Walter L.....750 Main St., Hartford, Conn., OALR
Hogshead, J. M.,
602 Georgia Ave., Chattanooga, Tenn., OALR
Hogue, D. W.....Fairbanks Bldg., Springfield, O., OALR
Hogue, G. I.....410 Jefferson St., Milwaukee, Wis., OALR
Hohf, J. A.....Clinic Bldg., Yankton, S. D., OALR
Holdsworth, Frank,
206 Wilhelm Blk., Traverse City, Mich., OALR
*Hollis, W. A...214 N. High St., Hartford City, Ind., OALR
Holloway, T. B..1819 Chestnut St., Philadelphia, Pa., Op.
Holmes, Benj. H.....432 Baker Bl., Racine, Wis., OALR
Holt, E. E., Jr.....823 Congress St., Portland, Me., Op.A
*Hompes, J. J...Security Mutual Bldg., Lincoln, Neb., OALR
Hood, T. C...Hume-Mansur Bldg., Indianapolis, Ind., Op.
Hopkins, W. E..515 Park Ave., New York City, N. Y., OALR
Hopper, Wilbur L..209 S. Main St., Fort Scott, Kan., OALR
Hotchkiss, W. W..195 Forest Ave., Jamestown, N. Y., OALR
Hourn, George E.,
703 University Club Bldg., St. Louis, Mo., ALR
Hovde, A. G....Board of Trade Bldg., Superior, Wis., OALR
*Howard, C. N.....Warsaw Clinic, Warsaw, Ind., OALR
Howard, E. F.First Natl. Bank Bldg., Vicksburg, Miss., ALR
Howard, Edward L..First Natl. Bk., Ft. Worth, Tex., OALR
Howard, J. W.....Rialto Bldg., Kansas City, Mo., OALR
Howard, Lloyd G.....Council Bluffs, Iowa, OALR
Howard, William H.,
137 N. Fairview Ave., St. Paul, Minn., OALR
Howe, Archibald W.,
Puget Sound Bk. Bldg., Tacoma, Wash., OALR
Hubbard, A. E.....372 Franklin St., Buffalo, N. Y., Op.
*Hughes, Thomas E.,
Medical Arts Bldg., Richmond, Va., ALR
Hughes, W. F.Hume-Mansur Bldg., Indianapolis, Ind., Op.
Huntington, Robt. H.....Eureka Springs, Ark., OALR
Hurd, L. M.....39 E. 50th St., New York City, N. Y., ALR
*Hurff, J. W.....86 Washington St., Newark, N. J., OALR
*Hurley, E. D.....419 Boylston St., Boston, Mass., Op.
Hurley, E. H.....419 Boylston St., Boston, Mass., Op.
Hurst, V. Reeves.....Longview, Tex., OALR
Husik, David N....1610 Spruce St., Philadelphia, Pa., ALR
Hussey, E. J.....276 High St., Holyoke, Mass., Op.LR

HUTCHINSON, O. G....Schweiter Bldg., Wichita, Kas., OALR

IGLAUER, S.....Livingston Bldg., Cincinnati, O., ALR
IMPERATORI, C. J..17 E. 38th St., New York City, N. Y., ALR
INGERMAN, S. M.,
418 Central Park, W., New York City, N. Y., OALR
INGERSOLL, J. M.....Osborn Bldg., Cleveland, O., ALR
INGLIS, H. J.....43 Bay State Bldg., Boston, Mass., ALR
INGRAM, L. C. M.....Clinic Bldg., Olanda, Fla., OALR
IRVIN, EDGAR H..321 W. Rio Grande St., El Paso, Tex., OALR
IRVINE, A. R.,
709 Westlake Professional Bldg., Los Angeles, Cal., Op.
IRWIN, F. N.....114 E. 54th St., New York City, N. Y., OALR
IRWIN, V. J.....389 Main St., Springfield, Mass., OALR
*IRWIN, VINCENT J., JR.389 Main St., Springfield, Mass., OALR
ISAACS, D.....Brandeis Theater Bldg., Omaha, Neb., OALR
ISRAEL, J. P.U. S. Veterans Bureau, San Francisco, Cal., OALR
*ISRAEL, N. E.....404 Carter Bldg., Houston, Tex., OALR
*ISRAEL, S.....Carter Bldg., Houston, Tex., OALR
IVINS, H. M.....408 Second Ave., Cedar Rapids, Ia., OALR

JACKLE, ARTHUR F...63 E. Fourth St., Dunkirk, N. Y., Op.
JACKSON, C.....128 S. 10th St., Philadelphia, Pa., L
*JACKSON, E.....Imperial Bldg., Denver, Colo., Op.
JACKSON, J. D.....2324 West 8th St., Erie, Pa.
*JACOBI, F.....Colton Bldg., Toledo, O., OALR
JACOBS, M. W.....Carleton Bldg., St. Louis, Mo., Op.
JACOBS, S. M...1018 E. 163rd St., New York City, N. Y., OALR
JACOBY, D. P. A.....106 Touro St., Newport, R. I., Op.AL
JAMES, J. H..Natl. City Bank Bldg., Mankato, Minn., OALR
JARECKY, H...138 W. 86th St., New York City, N. Y., OALR
*JEAN, GEORGE W.,
San Marcos Bldg., Santa Barbara, Cal., Op.
JENNINGS, J. E.....Carleton Bldg., St. Louis, Mo., OALR
*JESSAMAN, L. W.182 Concord St., Framingham, Mass., OALR
JOBSON, G. B.....4 Printz Bldg., Franklin, Pa., OALR
*JOHNSON, A. E..Citizens Bk. Bldg., Watertown, S. D., OALR
JOHNSON, H. R...512 Walnut Ave., Fairmont, W. Va., OALR
JOHNSON, OTIS H.,
630 Volunteer Bldg., Chattanooga, Tenn., OALR
JOHNSON, THOMAS H.,
30 W. 59th St., New York City, N. Y., Op.
*JOHNSTON, W. H.....Layrel Bldg., Muscatine, Ia., OALR
JOHNSTON, WILSON.....Stevens Bldg., Portland, Ore., OALR
JONES, CHARLES C.....19 Garfield Pl., Cincinnati, O., ALR
*JONES, E. L..First Natl. Bank Bldg., Cumberland, Md., OALR
JONES, E. W.....44 Atlanta St., Lynn, Mass., Op.A
JONES, F. W.....Times Bldg., Alton, Ill., OALR
JORDAN, G. T.....30 N. Michigan Blvd., Chicago, Ill., OALR
JOYES, CRITTENTON,
Farmers & Merch. Natl. Bk. Bldg., Ft. Worth, Tex., OALR

JOYNT, ALBERT J.....Black Bldg., Waterloo, Iowa, OALR
JUDD, HAROLD B..47 E. 57th St., New York City, N. Y., ALR
JUDGE, HARRY V.....228 State St., Albany, N. Y., Op.

*KAHN, HARRY.....104 S. Michigan Blvd., Chicago, Ill., ALR
KAHN, MYRON E.....29 E. Madison St., Chicago, Ill., ALR
*KAUFFMAN, ARNOLD B.,
305 Gurney Bldg., Syracuse, N. Y., ALR
KAUFMAN, A. S.....1923 Spruce St., Philadelphia, Pa., ALR
KEELER, J. C.....254 S. 16th St., Philadelphia, Pa., ALR
*KEIPER, G. F.....14 N. Sixth St., Lafayette, Ind., OALR
KEITH, D. M.....414 N. Main St., Rockford, Ill., ALR
*KEITH, W. E..807 Waldheim Bldg., Kansas City, Mo., OALR
*KEARBY, D. O.,
422 Am. Cen. Life Bldg., Indianapolis, Ind., ALR
*KEIM, WILLIAM F.....2 Lombardy St., Newark, N. J., OALR
KELLER, T. F.....The Spitzer, Toledo, O., OALR
KELLEY, C. W., JR..328 Francis Bldg., Louisville, Ky., OALR
KEMLER, JOSEPH J...1908 Eutaw Pl., Baltimore, Md., OALR
KENDALL, M. R.....614 Osborn Bldg., Cleveland, O., ALR
KENNEY, WM. L..Corby Forsee Bldg., St. Joseph, Mo., OALR
KENNON, WILLIAM G.Lambuth Bldg., Nashville, Tenn., OALR
KERRICK, STANLEY E.,
407 Security Bldg., Hollywood, Cal., OALR
KERRISON, P. D...58 W. 56th St., New York City, N. Y., ALR
KEY, B. W.....7 W. 49th St., New York City, N. Y., Op.
*KEYS, MORTON J.,
307 Jones Block, Victoria, B. C., Canada, OALR
KIEFER, H. A..406 Brockman Bldg., Los Angeles, Cal., OALR
*KIEHLE, FRED'K A..1000 Corbett Bldg., Portland, Ore., OALR
KILBOURNE, PERLEY H.Fidelity Med. Bldg., Dayton, O., OALR
KIMBERLIN, J. W...900 Rialto Bldg., Kansas City, Mo., Op.
KINCAID, J. H.....Ross Flats, Knoxville, Tenn., OALR
KING, C.....Union Central Bldg., Cincinnati, O., Op.
KING, CHARLES R.....471 Spitzer Bldg., Toledo, O., OALR
KING, EDWARD.....605 Traction Bldg., Cincinnati, O., ALR
KING, G. I.....537 E. Market St., Alliance, O., OALR
*KING, J. J.....40 E. 41st St., New York City, N. Y., ALR
KING, O. H...Dugan Stuart Bldg., Hot Springs, Ark., OALR
KIRBY, DANIEL B.30 W. 59th St., New York City, N. Y., Op.
KIRKENDALL, J. S.....315 N. Aurora St., Ithaca, N. Y., OALR
KIRKPATRICK, S.....Lauderdale Ave., Selma, Ala., OALR
KISER, J. D..Central Natl. Bk. Bldg., Bartlesville, Okla., OALR
*KISTLER, O. F..43 N. Franklin St., Wilkes-Barre, Pa., Op.AR
KISTNER, F. B.....Stevens Bldg., Portland, Ore., ALR
KITTLESON, T. N.101 Lincoln Ave., Fergus Falls, Minn., OALR
KLEENE, F.....1445 Wicker Park Ave., Chicago, Ill., OALR
KLINE, H. G.....University Blk., Syracuse, N. Y., ALR
KLINEDIENST, J. F.....220 S. George St., York, Pa., OALR
KLINGER, THOS. O..314 Landers Bldg., Springfield, Mo., OALR

KNAPP, A.....10 E. 54th St., New York City, N. Y., Op.
KNAPP, B...Intermediate Life Bldg., Evansville, Ind., OALR
*KNEEDLER, G. C.....Jenkins Bldg., Pittsburgh, Pa.
KNODE, A. R..Omaha Natl. Bank Bldg., Omaha, Neb., ALR
KOLLOCK, C. W.....86 Wentworth, Charleston, S. C., OALR
KOOBS, HERMAN J. G.4th and Walnut Sts., Rogers, Ark., OALR
KOPETZKY, S. J..51 W. 73rd St., New York City, N. Y., ALR
KRAFT, O. H.....25 E. Washington St., Chicago, Ill., Op.
KRAUSS, F.....1701 Chestnut St., Philadelphia, Pa., OALR
KREBS, A.....Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
KRENTZER, A. G.,
Trust Natl. Bank Bldg., Milwaukee, Wis., ALR
KRESS, G. H...245 Bradbury Bldg., Los Angeles, Cal., OALR
KRIEGER, G. M.....119 E. 6th St., Michigan City, Ind., OALR
*KRIEGER, WM. A..35 Market St., Poughkeepsie, N. Y., OALR
KRUG, E. F.....12 W. 44th St., New York City, N. Y., OALR
KULLY, B. M..631 City Natl. Bk. Bldg., Omaha, Neb., ALR

LAESSLE, HENRY A.,
The Lennox, 13th & Spruce Sts., Philadelphia, Pa., ALR
LAFORCE, B. D.....Hofman Bldg., Ottumwa, Ia., OALR
LA FORCE, E. F.....Tama Bldg., Burlington, Ia., OALR
LAMB, F. W.....7th and Vine Sts., Cincinnati, O., OALR
LAMB, HARVEY..826 Metropolitan Bldg., St. Louis, Mo., Op.
*LAMB, R. S.....Stoneleigh Ct., Washington, D. C., Op.
*LANCASTER, W. B.,
520 Commonwealth Ave., Boston, Mass., Op.
LANCASTER, WILSON.310 Dakota Ave., Wahpeton, N. D., OALR
LANDES, GEORGE A...110½ S. 18th St., Parsons, Kan., OALR
LANDFRIED, C. J.,
607 Land Title & Guarantee Bldg., New Orleans, La., ALR
LANGWORTHY, H. G...10th & Bluff Sts., Dubuque, Ia., OALR
LANING, J. H...726 Lathrop Bldg., Kansas City, Mo., ALR
LAPSLEY, ROBERT M.....Masonic Temple, Keokuk, Ia., OALR
*LARGE, S. H.....2323 Prospect Ave., Cleveland, O., ALR
*LARKIN, B. J..Hume-Mansur Bldg., Indianapolis, Ind., Op.
LARSEN, C. L.....Lowry Bldg., St. Paul, Minn., OALR
*LARUE, C. L.....17 Physicians Bldg., Boulder, Colo., OALR
*LASSALLE, ALBERT..493 St. Denis St., Montreal, Canada, OALR
LASSEN, F.....First Natl. Bank Bldg., Pueblo, Colo., ALR
LA VIGNE, A. A..11 E. 48th St., New York City, N. Y., OALR
LAWRENCE, G. A.Medical Arts Bldg., Philadelphia, Pa., Op.
LEA, JOS. A.....1125 Rialto Bldg., Kansas City, Mo., ALR
LEAVY, C. A.....Metropolitan Bldg., St. Louis, Mo., ALR
*LEDERMAN, I. A.....Atherton Bldg., Louisville, Ky., OALR
LEE, JOHN W...804 Besse Bldg., Minneapolis, Minn., OALR
*LEECH, J. W.....111 Broad St., Providence, R. I., OALR
LEFLER, ANNA B.,
605 W. P. Story Bldg., Los Angeles, Cal., OALR
LEHAN, J. W.....Park Pl. Bldg., Greeley, Colo., OALR

LEMERE, H. B.....Brandeis Bldg., Omaha, Neb., OALR
LEMOINE, A. N.....Lathrop Bldg., Kansas City, Mo., Op.
LENKER, JOHN N.....Osborn Bldg., Cleveland, O., ALR
LENT, E. J.....122 N. Lafayette St., South Bend, Ind., OALR
LEONARD, ARTHUR C.,
505 Bryant Bldg., Kansas City, Mo., OALR
*LERNER, MACY L.....332 Park Ave., Rochester, N. Y., Op.
LEVY, L...Bank of Commerce Bldg., Memphis, Tenn., OALR
LEVY, R.....Metropolitan Bldg., Denver, Colo., ALR
LEWIS, A. C.....Exchange Bldg., Memphis, Tenn., OALR
LEWIS, FIELDING O..259 S. 17th St., Philadelphia, Pa., ALR
LEWIS, F. P.....454 Franklin St., Buffalo, N. Y., Op.
LEWIS, GEORGE A.....29 Main St., Roundup, Mont., OALR
LEWIS, J. D....516 LaSalle Bldg., Minneapolis, Minn., OALR
LEWIS, J. P.....543 Boylston St., Boston, Mass., ALR
LEWIS, WILLIAM W.836 Lowry Bldg., St. Paul, Minn., OALR
LEWITT, F. C.....135 Stockton St., San Francisco, Cal., OAL
LIBBY, GEORGE F.....Majestic Bldg., Denver, Colo., Op.
*LICHTENBERG, J. S.....Rialto Bldg., Kansas City, Mo., Op.
LIDIKAY, CHAS. J.,
519 Portsmouth Bldg., Kansas City, Kans., OALR
LILLIE, HAROLD I.....Mayo Clinic, Rochester, Minn., ALR
LINDSAY, D. M...Boston Bldg., Salt Lake City, Utah, OALR
LINN, E. G.....Fleming Bldg., Des Moines, Ia., OALR
LINN, JAY G.....820 Highland Bldg., Pittsburgh, Pa., OALR
*LINTLEMAN, F. R....312 Hayes Blk., Janesville, Wis., OALR
*LISCHKOFF, M. A.....Blount Bldg., Pensacola, Fla., OALR
LITCHFIELD, J. T..609 Yeats Bldg., Minneapolis, Minn., OALR
LLOYD, A. W...504 Hammond Bldg., Hammond, Ind., OALR
LOEB, C.....25 E. Washington St., Chicago, Ill., Op.
LOEB, H. W.....537 N. Grand Ave., St. Louis, Mo., ALR
LOFTUS, J. E..605 Medical Arts Bldg., Philadelphia, Pa., ALR
LOKEY, H. M.....41 Doctors Bldg., Atlanta, Ga., OALR
LOMBARDO, MELCHIORE,
186 Meserole St., Brooklyn, N. Y., Op.
LOOMIS, E. A...Donaldson Bldg., Minneapolis, Minn., OALR
*LOOPER, EDWARD A.....605 Park Ave., Baltimore, Md., OALR
LORIE, A.....Commerce Bldg., Kansas City, Mo., ALR
LORING, J. BROWN..25 E. Washington St., Chicago, Ill., OALR
LOUCHERY, D. C..214 W. Main St., Clarksburg, W. Va., OALR
LOVE, JOHN G.....Ilgenfritz Bldg., Sedalia, Mo., OALR
LUBMAN, MAX..135 E. 65th St., New York City, N. Y., ALR
LUEDDE, W. H....Metropolitan Bldg., St. Louis, Mo., Op.
LUKENS, CHARLES.....218 Michigan St., Toledo, O., OALR
*LUKENS, ROBT, McD.,
1308 Hunting Park Ave., Philadelphia, Pa., ALR
LYMAN, HARRY W.....Carleton Bldg., St. Louis, Mo., ALR
LYNCH, R. C.,
632 Maison Blanche Bldg., New Orleans, La., ALR

LYONS, HORACE R...30 N. Michigan Blvd., Chicago, Ill., ALR

MACFARLANE, P. B.152 St. James St., S., Hamilton, Can., OALR
MACFARLIN, D...1805 Chestnut St., Philadelphia, Pa., OALR
MACINE, J. S..503 Donaldson Bldg., Minneapolis, Minn., OALR
MACKENZIE, ALICE V.1724 Spruce St., Philadelphia, Pa., OALR
*MACKENZIE, GEORGE W.,
1724 Spruce St., Philadelphia, Pa., OALR
*MAC LACHLAN, A. A.....May Bldg., Pittsburgh, Pa., ALR
MADDEN, REED.....Allen Bldg., Xenia, O., OALR
MAGEE, R. S.....Mills Bldg., Topeka, Kan., Op.A
MAGGARD, DELANO I.....Beacon Bldg., Wichita, Kan., OALR
MAGRUDER, A. C..Burns Bldg., Colorado Springs, Colo., OALR
MAIDEN, S. D...217 Merriam Blk., Council Bluffs, Ia., OALR
MAIRE, L. E.....Park Bldg., Detroit, Mich., Op.A
MALONEY, THOS. J..1032 Lowry Bldg., St. Paul, Minn., OALR
*MANN, R. H. T..State Natl. Bk. Bldg., Texarkana, Tex., OALR
MANSUR, D. W...Brockman Bldg., Los Angeles, Cal., Op.
*MARCH, CLARA A....465 Ashland Ave., Buffalo, N. Y., Op.
*MARKEL, J. C....Westinghouse Bldg., Pittsburgh, Pa., Op.
MARQUIS, G. P.....30 N. Michigan Blvd., Chicago, Ill., ALR
*MARSHALL, GEORGE G....Gryphon Bldg., Rutland, Vt., OALR
MARSHALL, W..Diamond Bank Bldg., Pittsburgh, Pa., ALR
MARTIN, ANCIL....207 Goodrich Bldg., Phoenix, Ariz., Op.A
MARTIN, H. H.....247 Bull St., Savannah, Ga., OALR
MATHENY, R. C.....306 E. Main St., Galesburg, Ill., OALR
MATHESON, J. P.,
511 Independence Bldg., Charlotte, N. C., OALR
MATTHEWS, J..Metropolitan Bldg., Minneapolis, Minn., LR
MAULDIN, L. O.....Wallace Bldg., Greenville, S. C., OALR
MAUMENEE, A. E.....156 S. Francis St., Mobile, Ala., OALR
MAXEY, E. E.....4 Electric Bldg., Aberdeen, Wash., OALR
MAY, C. H...698 Madison Ave., New York City, N. Y., Op.
MAY, J. V.....First Natl. Bank Bldg., Marinette, Wis., OALR
MAY, JAMES W..800 Minnesota Ave., Kansas City, Mo., Op.
MAYBAUM, J. L...12 E. 86th St., New York City, N. Y., ALR
MEANOR, WM. C.,
619 Westinghouse Bldg., Pittsburgh, Pa., Op.
MEANS, C. S.,
1006 Pacific-Southwest Bldg., Long Beach, Cal., OALR
MEIERHOF, E. L.,
1140 Madison Ave., New York City, N. Y., OALR
MERCER, RAY.....Stein Bldg., Quincy, Ill., OALR
*MERCER, R. E...David Whitney Bldg., Detroit, Mich., LR
MERRILL, H. G.....35 East, 2 South St., Provo, Utah, OALR
MERRILL, W. H..The Bay State Bldg., Lawrence, Mass., OALR
MESSINGER, H. C.....170 Broad St., Providence, R. I., Op.
METZ, R. B.....Guardian Bldg., Cleveland, O., Op.
METZENBAUM, M.....Rose Bldg., Cleveland, O., ALR
MIDDLETON, A. B.....201 Sterry Blk., Pontiac, Ill., OALR

MIDDLETON, HARRY E.....Times Bldg., Alton, Ill., OALR
MILLER, ALICE R.....West Palm Beach, Fla., OALR
MILLER, C. E.....315 S. Jefferson St., Muncie, Ind., OALR
MILLER, C. M..Stuart Circle Hospital, Richmond, Va., OALR
*MILLER, E. B.....2008 Walnut St., Philadelphia, Pa., Op.
MILLER, HUGH..520 Chambers Bldg., Kansas City, Mo., OALR
MILLETTE, J. W.....Reibold Bldg., Dayton, O., OALR
MINER, S. C.....1564 Woodward Ave., Detroit, Mich., ALR
*MINOR, C. L.....Fairbanks Bldg., Springfield, O., OALR
MINOR, H. F.....Randolph Bldg., Memphis, Tenn., OALR
MINTON, WM. H.....Bartlett Bldg., St. Joseph, Mo., OALR
MITCHELL, E. K.,
710 W. Lehigh Ave., Philadelphia, Pa., ALR
MITHOEFER, W.....19 Garfield Pl., Cincinnati, O., ALR
MITTENDORF, A. D.399 Park Ave., New York City, N. Y., Op.
MITTENDORF, W. K.,
115 E. 53rd St., New York City, N. Y., Op.A
MOISE, A. B.....Shreveport, La., ALR
MOLT, W. F..Newton Claypool Bldg., Indianapolis, Ind., ALR
MONAGHAN, D. G...535 Majestic Bldg., Denver, Colo., Op.
MONAHAN, J. A.....181 Chestnut St., Clinton, Mass., OALR
MONOSMITH, O. B.....424 Broadway, Lorain, O., OALR
MONSON, S. H.....Ainsfield Bldg., Cleveland, O., OALR
*MOORE, G. A.....Bank Bldg., Palmer, Mass., OALR
*MOORE, GEORGE H...200 Main St., Schuylkill Haven, Pa., OALR
MOORE, M.....The Rochambeau, Washington, D. C., ALR
*MOORE, T. K.....416 Hamilton Bldg., Akron, O., OALR
*MOORE, T. W...1050 Fifth Ave., Huntington, W. Va., OALR
*MOORE, W. F.....255 S. 16th St., Philadelphia, Pa., ALR
MORGAN, J. A....Young Bldg., Honolulu, T. Hawaii, OALR
*MORRIS, A. G.....41 Gibbs St., Rochester, N. Y., OALR
MORRISON, F. A..Willoughby Bldg., Indianapolis, Ind., Op.
MORSE, A. W.....507 Phoenix Bldg., Butte, Mont., OALR
MORSE, J. A..503 Donaldson Bldg., Minneapolis, Minn., OALR
*MORSMAN, L. W..203 Congdon Bldg., Hibbing, Minn., OALR
MORTON, HOWARD MCI.,
Metropolitan Bk. Bldg., Minneapolis, Minn., OALR
*MORTON, JOHN B..90 E. Warren Ave., Detroit, Mich., OALR
*MOSHER, H. P.....828 Beacon St., Boston, Mass., ALR
MOSS, HOWARD E..835 Rialto Bldg., Kansas City, Mo., OALR
MOSS, R. E.....Hicks Bldg., San Antonio, Tex., OALR
MOTT, JOHN S.....214 Argyle Bldg., Kansas City, Mo., OALR
MOULTON, EVERETT C.,
Merchants Natl. Bank Bldg., Ft. Smith, Ark., OALR
MOULTON, H.,
Merchants Natl. Bank Bldg., Ft. Smith, Ark., OALR
MULLIN, W. V..Burns Bldg., Colorado Springs, Colo., ALR
MULSOW, JOHN E., 410 Jefferson St., Milwaukee, Wis., OALR
MUNCASTER, S. B.....921, 15 A St., Washington, D. C., Op.A
MUNCY, W. M...23 Waterman St., Providence, R. I., OALR

MUNDT, G. H....25 E. Washington St., Chicago, Ill., OALR
MURDOCH, J. F.....Bessemer Bldg., Pittsburgh, Pa., Op.
MURPHY, J. G...Murchison Bldg., Wilmington, N. C., OALR
MURPHY, W. E.....7th and Race Sts., Cincinnati, O., OALR
MURRAY, ALFRED N...4654 Sheridan Rd., Chicago, Ill., OALR
*MURRAY, WM. R...Nicollet Clinic, Minneapolis, Minn., OALR
*MUSSUN, WILLIAM G.....Osborn Bldg., Cleveland, O., ALR
*MYERS, ELMER A.,
407 Board of Trade Bldg., Superior, Wis., ALR
MYERS, JOHN L..626 Lathrop Bldg., Kansas City, Mo., OALR
MYERSON, M. C.....184 Clinton St., Brooklyn, N. Y., ALR
MCALESTER, ANDREW W., JR.,
625 Bryant Bldg., Kansas City, Mo., Op.
*MCALISTER, J. C.....10 S. Mill Ave., Ridgeway, Pa., OALR
MCBEAN, GEORGE M.,
22 E. Washington St., Chicago, Ill., OALR
MCBRIDE, W. O.,
218 Washington Blvd., Ft. Wayne, Ind., OALR
MCCANNELL, ARCHIBALD D.,
Colcord Blk., Minot, N. D., OALR
MCCARTNEY, OLIVER P.,
314 Portsmouth Bldg., Kansas City, Kans., OALR
*MCCASKEY, C. H.,
Amer. Cent. Life Bldg., Indianapolis, Ind., ALR
MCCAW, JOHN A....418 Majestic Bldg., Denver, Colo., Op.
MCCLELLAND, L. A.,
78 McDonough St., Brooklyn, N. Y., ALR
MCCLELLAND, CARL C.,
David Whitney Bldg., Detroit, Mich., OALR
MCCONNELL, T. E.,
Alter Bldg., New Kensington, Pa., OALR
*MCCOOL, J. L.....Stevens Bldg., Portland, Ore., Op.
MCCORTY, VIRGIL W.,
800 Rialto Bldg., Kansas City, Mo., OALR
MCCOY, J.....157 W. 73rd St., New York City, N. Y., ALR
MCCREADY, J. H.....Empire Bldg., Pittsburgh, Pa., ALR
*MCCULLOUGH, R. J. P..102 College St., Toronto, Can., OALR
*MCDERMOTT, JOHN J.,
St. Joseph Sanitarium, St. Joseph, Mich., OALR
MCDIARMID, H. O.,
Rossner Ave. and 9th St., Brandon, Manitoba, Can., OALR
MCDONALD, C. L.....432 Ainsfield Bldg., Cleveland, O., OALR
*MCDOWELL, N. D., 275 Alexander St., Rochester, N. Y., OALR
MCFARLANE, MURRAY,
190 Bloor St., E., Toronto, Can., OALR
*MCGINNIS, EDWIN,
104 S. Michigan Blvd., Chicago, Ill., ALR
MCGIVERN, CHAS. S.,
805 Pacific Ave., Atlantic City, N. J., ALR
*MCGUIRE, H. H..105 N. Braddock St., Winchester, Va., OALR

*McHenry, D. D.,
301 Colcord Bldg., Oklahoma City, Okla., OALR
*McKee, George J.,
1118 Westinghouse Bldg., Pittsburgh, Pa., ALR
*McKee, S. H.....158 Crescent St., Montreal, Can., Op.
*McKelvey, A. D. M.....160 Bloor St., Toronto, Can., ALR
McKennan, J. W.,
Washington Trust Bldg., Washington, Pa., Op.
*McKeown, E. E...Metropolitan Bldg., Denver, Colo., OALR
*McKinney, Alexander R.,
Bearinger Bldg., Saginaw, Mich., OALR
McKinney, R.,
Bk. of Commerce & Trust Bldg., Memphis, Tenn., ALR
McLaughlin, P. A.,
73 S. Washington St., Wilkes-Barre, Pa., OALR
*McMurray, J. B.,
Washington Trust Bldg., Washington, Pa., OALR
McNaught, H.....Butler Bldg., San Francisco, Cal., ALR
McReynolds, G. S....State Bank Bldg., Temple, Tex., OALR
McReynolds, J. O.....Trust Bldg., Dallas, Tex., OALR

Nadeau, E. G.,
Bellin-Buchanan Bldg., Green Bay, Wis., OALR
Naftzger, J. B.....Davidson Bldg., Sioux City, Ia., OALR
Nance, W. O.....30 N. Michigan Blvd., Chicago, Ill., OALR
Neeper, E. R.,
Ex. Natl. Bank Bldg., Colorado Springs, Colo., Op.
Neighbors, Clarence A.,
Underwood Bldg., Ottawa, Kan., OALR
New, Gordon B.....Mayo Clinic, Rochester, Minn., LR
Nelson, C. F.....Schofield Bldg., Cleveland, O., OALR
Nelson, L. A.....Lowry Arcade, St. Paul, Minn., Op.A
Nelson, Lloyd L.....Norfolk, Neb., OALR
Newell, W. S.,
1029 Vermont Ave., Washington, D. C.
*Newhart, H.....Donaldson Bldg., Minneapolis, Minn., OALR
Newton, L. A.,
317 Colcord Bldg., Oklahoma City, Okla., OALR
Nisselson, Max,
616 Madison Ave., New York City, N. Y., OALR
Norcross, Edward P., 30 N. Michigan Ave., Chicago, Ill., ALR
Norton, Roy R...Masonic Bldg., New Brighton, Pa., OALR
Novak, Frank, Jr.,
30 N. Michigan Blvd., Chicago, Ill., ALR
Noyes, G. L.....311 Hitt St., Columbia, Mo., OALR
Nutter, C. F.....16 Amherst St., Nashua, N. H., Op.

O'Brien, C. S.....Colton Bldg., Toledo, Ohio, Op.
*O'Brien, S. A.,
400 1st Natl. Bank Bldg., Mason City, Ia., OALR

OBERG, C. M.....Syndicate Bldg., Minneapolis, Minn., OALR
O'CONNOR, D. F.....671 Broad St., Newark, N. J., OALR
O'CONNOR, R. P.....1206 Broadway, Oakland, Cal., Op.
*ODELL, A.....32 Adams St., Detroit, Mich., Op.AL
O'DELL, TIMOTHY...112A Madison Ave., Aurora, Mo., OALR
OERTEL, T. E.....638 Green St., Augusta, Ga., OALR
OESTERLING, H. E.,
Wheeling Steel Corp. Bldg., Wheeling, W. Va., OALR
OFFUTT, W. N.....Security Bldg., Lexington, Ky., OALR
OLSHO, SYDNEY L....235 S. 15th St., Philadelphia, Pa., OALR
OPPENHEIMER, S.,
45 E. 60th St., New York City, N. Y., OALR
*ORCUT, D. C.....7 W. Madison St., Chicago, Ill., Op.A
O'REILLY, C. A...1901 Chestnut St., Philadelphia, Pa., ALR
O'REILLY, W. F.....44 S. Common St., Lynn, Mass., OALR
*ORTON, H. B.....671 Broad St., Newark, N. J., ALR
OVERMAN, F. V.,
Hume-Mansur Bldg., Indianapolis, Ind., ALR
OWEN, F. S.....Brandeis Bldg., Omaha, Neb., OALR

PAGE, L.....Hume-Mansur Bldg., Indianapolis, Ind., ALR
PAINTER, A. M.....Sharp Bldg., Kansas City, Mo., ALR
PALMER, EDW. M....629 Beacon Bldg., Wichita, Kan., OALR
PARK, J. W.....32 N. Second St., Harrisburg, Pa., OALR
PARKER, E. H....La Salle Bldg., Minneapolis, Minn., LR
*PARKER, W. R...David Whitney Bldg., Detroit, Mich., Op.
PARRISH, MAJ. ROBERT E., M. C., U. S. A.,
Walter Reed Gen. Hospital, Washington, D. C., ALR
PARSONS, J. G.....Paulton Bldg., Sioux Falls, S. D., OALR
PATTERSON, E. J.,
Westinghouse Bldg., Pittsburgh, Pa., ALR
PATTERSON, J. A.,
Burns Bldg., Colorado Springs, Colo., OALR
PATTERSON, JOSEPH M.,
518 Bryant Bldg., Kansas City, Mo., OALR
PATTERSON, W. E.,
808 La Salle Bldg., Minneapolis, Minn., OALR
PATTON, J. M.....Brandeis Bldg., Omaha, Neb., OALR
PAYNE, A. L.....Opera House Blk., Eau Claire, Wis., OALR
PEARLMAN, SAMUEL J..29 E. Madison St., Chicago, Ill., ALR
*PEARSON, W. W..Bankers Trust Bldg., Des Moines, Ia., OALR
PEIRCE, HOWARD W.,
David Whitney Bldg., Detroit, Mich., ALR
PEELER, C. N.....W. 7th St., Charlotte, N. C., OALR
*PETER, LUTHER C.,
N. E. cor. Chestnut & 20th Sts., Philadelphia, Pa., Op.
PETERMAN, H. E....518 N. Charles St., Baltimore, Md., OALR
PFAFFLIN, C. A.,
Newton Claypool Bldg., Indianapolis, Ind., OALR

PFINGST, A. O.....Atherton Bldg., Louisville, Ky., Op.
PFINGSTEN, C. F....508 N. Grand Ave., St. Louis, Mo., ALR
PHELPS, KENNETH A.,
730 La Salle Bldg., Minneapolis, Minn., OALR
PHILLIPS, W. C.....40 W. 47th St., New York City, ALR
PHILLIPS, W. L...759 Richmond Ave., Buffalo, N. Y., Op.
PHINNEY, F. D.....22 W. 7th St., Cincinnati, O., OALR
PITKIN, C. E.....Osborn Bldg., Cleveland, O., ALR
*POLLOCK, H. L.....2551 N. Clark St., Chicago, Ill., ALR
PONTIUS, P. J....1831 Chestnut St., Philadelphia, Pa., OALR
*PORTER, E. H.....85 Madison St., Tiffin, O., OALR
PORTER, JESSE B.....415 S. 2nd St., Elkhart, Ind., OALR
*PORTER, L. B....117 Waterman St., Providence, R. I., OALR
POSEY, WM. CAMPBELL,
21st and Chestnut Sts., Philadelphia, Pa., Op.
POST, LAWRENCE,
520 Metropolitan Bldg., St. Louis, Mo., Op.
POST, MARTIN H.,
520 Metropolitan Bldg., St. Louis, Mo., Op.
POST, WINIFRED B....1380 S. Main St., Carthage, Mo., OALR
POTTER, GEORGE B.,
402 City Natl. Bank Bldg., Omaha, Neb., OALR
POTTER, L. F.....68 Walnut St., New Bedford, Mass., OALR
POTTER, PETER,
Granate and Alaska Sts., Butte, Mont., OALR
POTTS, J. B.....Brandeis Bldg., Omaha, Neb., Op.
POWERS, E...Central Natl. Bank Bldg., Carthage, Mo., OALR
PRANGEN, AVERY DE HART,
Mayo Clinic, Rochester, Minn., Op.
PRATT, F. J., JR.,
Metropolitan Bank Bldg., Minneapolis, Minn., OALR
*PRATT, J. A.,
Metropolitan Bank Bldg., Minneapolis, Minn., OALR
*PRENDERGAST, D.....1110 Euclid Ave., Cleveland, O., OALR
PRICE, N. W.....445 3rd St., Niagara Falls, N. Y., OALR
PROETZ, ARTHUR W.,
University Club Bldg., St. Louis, Mo., ALR
PROUD, WILLARD C.,
Phys. & Surg. Bldg., St. Joseph, Mo., OALR
PROUT, A. W.....370 E. Town St., Columbus, O., OALR
PUTNAM, F. I.,
708 Sioux Falls Natl. Bk. Bldg., Sioux Falls, S. D., OALR
PYLE, W...18th and Exchange Pl., Jersey City, N. J., OALR

QUINBY, WM. O'G.....14 James St., Newark, N. J., OALR
QUITTNER, S. S.....3912 Prospect Ave., Cleveland, O., OALR

RADCLIFFE, MCCLUNEY,
1906 Chestnut St., Philadelphia, Pa., Op.

RALLS, C. T.....1st Natl. Bank Bldg., Winfield, Kas., OALR
RALSTON, WALLACE W.,
1300 Walker Ave., Houston, Tex., Op.
RAVDIN, B. D.....223 Main, Evansville, Ind., OALR
*RAVDIN, M.....223 Main St., Evansville, Ind., OALR
RAY, V.....19 W. 7th St., Cincinnati, O., OALR
REAVES, CHAS. R.....Box No. 187, Knoxville, Tenn., OALR
REAVES, W. P.,
117 W. Sycamore St., Greensboro, N. C., OALR
REBUCK, C. S.....412 N. Third St., Harrisburg, Pa., OALR
RECTOR, A. E.....812 College Ave., Appleton, Wis., OALR
REEDER, J. E.....Davidson Bldg., Sioux City, Ia., OALR
REESE, R. G....50 W. 52nd St., New York City, N. Y., Op.
REESE, WARREN S...230 S. 21st St., Philadelphia, Pa., Op.
*REGER, H. S.,
New Wellman Bldg., Jamestown, N. Y., OALR
REICHE, O. C.....328 West Broad St., Hazleton, Pa., OALR
REIM, HUGO...417 Metropolitan Bldg., St. Louis, Mo., Op.
REMMENS, N.....2954 Logan Blvd., Chicago, Ill., Op.
RENAUD, G. L.....Fine Arts Bldg., Detroit, Mich., OALR
RENNER, W. S.....341 Linwood Ave., Buffalo, N. Y., ALR
REPASS, ROBERT E....150 Maple Rd., Indianapolis, Ind., ALR
REULING, F. HAROLD.....Black Bldg., Waterloo, Ia., OALR
REYNOLDS, H. G.....Natl. Bank Bldg., Paducah, Ky., Op.A
*REYNOLDS, J. S.,
816 La Salle Bldg., Minneapolis, Minn., OALR
RICE, C. P.....7 Sterns Bldg., Wahpeton, N. D., OALR
RICE, G. B...270 Commonwealth Ave., Boston, Mass., ALR
RICE, JOHN E.....390 Main St., Worcester, Mass., OALR
RICE, R. H.....Coshwell Bldg., Milwaukee, Wis., Op.AL
RICHARDS, LYMAN G.,
270 Commonwealth Ave., Boston, Mass., ALR
RICHARDSON, C. W.,
1317 Connecticut Ave., Washington, D. C., ALR
RICHARDSON, J. J.,
1509 Ave. of Presidents, Washington, D. C., ALR
RICHIE, L. T.....McCormick Bldg., Trinidad, Colo., OALR
RIDEOUT, W. J.....90 Stephenson St., Freeport, Ill., OALR
*RIDPATH, R. F....1928 Chestnut St., Philadelphia, Pa., ALR
*RIEGER, WALTER H.....Hanna Bldg., Cleveland, O., ALR
RINDLAUB, ELIZABETH P.,
DeLendrecie Block, Fargo, N. D., OALR
RINDLAUB, JOHN H.,
DeLendrecie Block, Fargo, N. D., OALR
RINDLAUB, MARTIN P., JR.,
DeLendrecie Block, Fargo, N. D., OALR
RINGLE, C. A.....Coronado Bldg., Greeley, Colo., ALR
RINGUEBERG, E. N. S.....15 Main St., Lockport, N. Y., Op.
*RISDON, ERNEST F....30 Avenue Rd., Toronto, Canada, ALR

DE RIVER, JOSEPH PAUL,
U. S. Veterans' Bureau, San Francisco, Calif., OALR
*ROBERTS, S. E.....Waldheim Bldg., Kansas City, Mo., ALR
ROBERTS, W. H...461 E. Colorado St., Pasadena, Cal., OALR
ROBERTSON, C. M.....30 N. Michigan, Chicago, Ill., OALR
ROBERTSON, E. N....132½ W. 6th St., Concordia, Kan., OALR
ROBERTSON, F. D..Ashton Bldg., Grand Rapids, Mich., OALR
*ROBINSON, H. T.....Medical Bldg., Cumberland, Md., OALR
ROBINSON, JAMES M....113 S. 6th St., Hiawatha, Kan., OALR
*ROBINSON, ROBERT E.,
205½ E. Bremer Ave., Waverly, Ia., OALR
ROGERS, A. S...105 S. Jefferson Ave., Saginaw, Mich., OALR
*ROGERS, J. T.,
758 Sherbrooke St., W., Montreal, Canada, ALR
ROLLER, L. A...122 Monroe St., Grand Rapids, Mich., OALR
ROMMEL, JOHN C.,
4501 N. Broad St., Philadelphia, Pa., OALR
ROOKER, A. L.,
1347 Michigan Ave., Niagara Falls, N. Y., ALR
ROOST, F. H.....515 Trimble Blk., Sioux City, Ia., OALR
*ROSENBAUM, J.....206 Bishop St., Montreal, Can., Op.
ROTH, ALBERT W.....303 Palace Bldg., Tulsa, Okla., OALR
ROTHSCHILD, HAROLD J.,
239 Lowry Bldg., St. Paul, Minn., OALR
ROTT, O. M.....Paulsen Bldg., Spokane, Wash., OALR
ROW, G. S....Hume-Mansur Bldg., Indianapolis, Ind., Op.
ROWLAND, J. F....Thompson Bldg., Hot Springs, Ark., OALR
*ROWLAND, WILLIAM D.,
220 Clarendon St., Boston, Mass., Op.
*RUBENTHAL, C.....Brandeis Bldg., Omaha, Neb., OALR
*RUBY, F. M.....Physicians Bldg., Union City, Ind., OALR
RUCKER, E. W., JR.,
926 Woodward Bldg., Birmingham, Ala., ALR
RUSH, CALVIN C.....342 Main St., Johnstown, Pa., ALR
RUSSELL, EDWIN L.,
402 Argyle Bldg., Kansas City, Mo., OALR
RUSSELL, J. A.....206 Masonic Temple, Erie, Pa., ALR
RUSSELL, RALPH E.....Waterloo, Iowa, OALR
RUST, E. G.....Hanna Bldg., Cleveland, O., Op.A
RYAN, LAWRENCE R.,
San Marcos Bldg., Santa Barbara, Calif., Op.A
RYDER, D. R.....151 Rock St., Fall River, Mass., ALR
SACCO, ANTHONY G.,
440 Clinton Ave., West Hoboken, N. J., OALR
*SALINGER, SAMUEL..25 E. Washington St., Chicago, Ill., ALR
SALLANDER, FREDERICK W.,
303 Security Bank Bldg., Sioux City, Ia., OALR
SANDERSON, D. D.....Funke Bldg., Lincoln, Neb., OALR
SANDERSON, H. H.,
David Whitney Bldg., Detroit, Mich., OALR

SARTAIN, PAUL J...2006 Walnut St., Philadelphia, Pa., Op.
SATTERLEE, R. H...187 Delaware Ave., Buffalo, N. Y., Op.
SAUER, W. E.....Humboldt Bldg., St. Louis, Mo., ALR
SAUER, W. W.....Box 416, Marietta, O., OALR
SAVAGE, G. H....Central Bank Bldg., Memphis, Tenn., OALR
SAVERY, C. E...126 N. Lafayette St., South Bend, Ind., OALR
SCALES, H. L.,
504-5 Rorabaugh-Wiley Bldg., Hutchinson, Kan., OALR
SCALES, J. L.,
Commercial Bank Bldg., Shreveport, La., OALR
SCALES, J. W.....202½ Main St., Pine Bluff, Ark., OALR
SCARLET, HUNTER W.,
2228 Locust St., Philadelphia, Pa., Op.
SCHAFFERT, N. L.,
57 S. Washington St., Wilkes-Barre, Pa., OALR
SCHATZ, HENRY A.,
2035 Chestnut St., Philadelphia, Pa., ALR
SCHENCK, CHARLES P.,
301 Texas St. Bk. Bldg., Fort Worth, Tex., OALR
*SCHILD, E. H...Cleveland Ave. and 9th St., Canton, O., Op.A
SCHILLER, A. NOAH,
225 West End Ave., New York City, N. Y., ALR
SCHLANSER, ADAM E.,
Walter Reed U. S. A. Gen. Hosp., Washington, D. C., OALR
SCHLEGEL, H. T.....501 Third St., Wausau, Wis., OALR
SCHLINDWEIN, G. W.....138 W. 9th St., Erie, Pa., OALR
SCHNEIDEMANN, T. B.,
Professional Bldg., Philadelphia, Pa., Op.
SCHOLZ, R. P.....Metropolitan Bldg., St. Louis, Mo., ALR
SCHRANT, J. H.,
504 Natl. Bank Bldg., Hutchinson, Kan., OALR
SCHURTER, M. A.....8 Morton Ave., Riverside, N. J., OALR
SCHUSTER, STEPHEN A.,
1st Natl. Bank Bldg., El Paso, Tex., ALR
SCHUTZ, W. H....712 Bryant Bldg., Kansas City, Mo., OALR
SCHWARTZ, FREDERICK A.,
601 Metropolitan Bldg., St. Louis, Mo., Op.
*SCHWEINITZ, GEO. E. DE,
1705 Walnut St., Philadelphia, Pa., Op.
SCHWENK, P. N. K.,
1417 N. Broad St., Philadelphia, Pa., Op.
SCOTT, JOHN R.....201 S. Main St., Ottawa, Kan., OALR
*SCROGGS, J. J.....218 E. Third St., Beaver, Pa., OALR
SEAMAN, GILBERT E.,
309 Goldsmith Bldg., Milwaukee, Wis., OALR
SEARCY, H. B...807 Greensboro Ave., Tuscaloosa, Ala., OALR
*SEARS, WM. H.....514 Penn St., Huntingdon, Pa., OALR
SEAY, D. E...Dallas Co. State Bk. Bldg., Dallas, Tex., OALR
SECOY, FRANK L.....525 Francis Bldg., Sioux City, Ia., OALR
SEDGWICK, O. W.....Ionia, Mich., OALR

SEDWICK, W. A.....Imperial Bldg., Denver, Colo., OALR
SEEGMAN, S.....Jenkins Arcade Bldg., Pittsburgh, Pa., ALR
SEELYE, WALTER K.,
806 American Bank Bldg., Seattle, Wash., OALR
SELFRIDGE, G. L....Schroth Bldg., San Francisco, Cal., ALR
SELLERS, LYLE M..430 Argyle Bldg., Kansas City, Mo., ALR
*SENER, WALTER J...130 So. 18th St., Philadelphia, Pa., OALR
SERNOFFSKY, ISAAC.....37 Allen St., Buffalo, N. Y., Op.
SEWALL, E. C.....Lane Hospital, San Francisco, Cal., OALR
SEYDELL, E. M.....201 N. Main St., Wichita, Kan., ALR
SHACKLETON, W. E.,
2323 Prospect Ave., Cleveland, O., Op.
SHAFER, J. J.....Stark Bldg., Louisville, Ky., OALR
SHAHAN, W. E....Metropolitan Bldg., St. Louis, Mo., Op.
*SHANAHAN, T. J.....419 Boylston St., Boston, Mass., ALR
SHANKLIN, E. M....575 Hohman St., Hammond, Ind., OALR
SHANNON, C. E.....104 Copley Rd., Upper Darby, Pa., Op.
SHAPERO, I. M.....365 East Ave., Rochester, N. Y., OALR
SHARRETT, GEORGE O.,
119 Bedford St., Cumberland, Md., OALR
SHASTID, T. H.....1920 Johns Ave., Superior, Wis., OALR
SHAWHAN, GLENN E.....Nampa, Ida., OALR
*SHEA, J. J.....Exchange Bldg., Memphis, Tenn., OALR
SHELLMAN, J. L.....Lowry Bldg., St. Paul, Minn., OALR
SHEMELEY, WM. G., JR.,
1724 Spruce St., Philadelphia, Pa., OALR
SHERMAN, MAJ. A. E.,
Army & Navy General Hosp., Hot Springs, Ark., OALR
SHERMAN, E. S.....Wiss Bldg., Newark, N. J., OALR
*SHIELDS, J. M.....Metropolitan Bldg., Denver, Colo., Op.
SHIELDS, LAWRENCE.....Allen Bldg., Xenia, O., ALR
SHIRAS, HOWARD H.,
629 Guardian Bldg., Cleveland, O., Op.
SHOEMAKER, FERDINAND,
728 Union St., Emporia, Kan., OALR
SHOEMAKER, S. A.,
U. S. Pub. Ser. Hosp., Kansas City, Mo., OALR
SHOEMAKER, W. A.....Carleton Bldg., St. Louis, Mo., Op.
SHORT, Z. N...Dugan Stuart Bldg., Hot Springs, Ark., OALR
SHREVE, O. M.....162 W. 8th St., Erie, Pa., OALR
SHULTS, EDWIN J. G....Haines Bldg., Eldorado, Kan., OALR
SHUMAN, G. H.....1105 Park Bldg., Pittsburgh, Pa., Op.
*SHUMATE, DAVID L.,
517 Shukert Bldg., Kansas City, Mo., OALR
SHURLY, B. R.....62 W. Adams St., Detroit, Mich., ALR
SHUTE, D. K.....1717 DeSales St., Washington, D. C., Op.
SIEGEL, F. X.,
2700 Union Central Bldg., Cincinnati, O., OALR
SILBERNAGEL, C. E.....283 E. State St., Columbus, O., OALR
SIMONDS, O. F.....Hanna Bldg., Cleveland, O., Op.

SIMPSON, H. LEE,
1413 David Whitney Bldg., Detroit, Mich., ALR
SIMPSON, JOHN R.,
1005 Westinghouse Bldg., Pittsburgh, Pa., ALR
SIMPSON, W. L.....Exchange Bldg., Memphis, Tenn., OALR
SINGLETON, E. M.,
111 E. Church St., Marshalltown, Ia., OALR
*SKILLERN, R. H...1928 Chestnut St., Philadelphia, Pa., LR
*SLEIGHT, R. D.....Post Bldg., Battle Creek, Mich., OALR
*SLOAN, HENRY L.,
Independence Bldg., Charlotte, N. C., Op.
*SLOCUM, G.....328 E. Huron St., Ann Arbor, Mich., Op.
SLUDER, G.....3542 Washington Ave., St. Louis, Mo., ALR
SMITH, ARTHUR E.,
1016 Donaldson Bldg., Minneapolis, Minn., OALR
SMITH, D.....834 Myrtle Ave., Bridgeport, Conn., Op.A
SMITH, D. S.....State Bank Bldg., La Crosse, Wis., OALR
SMITH, E. T.,
Connecticut Mut. Bldg., Hartford, Conn., Op.
SMITH, G. B.,
Broad St. and Fourth Ave., Rome, Ga., OALR
SMITH, H. A.....640 Main St., Delta, Colo., OALR
SMITH, J. M....134½ N. Patterson St., Valdosta, Ga., OALR
SMITH, R. C...Board of Trade Bldg., Superior, Wis., OALR
SMITH, S. McC....1420 Spruce St., Philadelphia, Pa., ALR
SMITH, S. S....Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
SMITH, V. C.,
Maison Blanche Bldg., New Orleans, La., Op.
SNEED, CARL M.....506 W. Broadway, Columbia, Mo., OALR
SNOW, L. W.,
Deseret Bank Bldg., Salt Lake City, Utah, OALR
SNYDER, W. H.....211 Ontario St., Toledo, O., OALR
SOMBERG, JOSEPH S....45 E. 60th St., New York, N. Y., Op.
SONNENSCHEIN, R.....Heyworth Bldg., Chicago, Ill., ALR
SPAKE, L. B.,
522 Minnesota Ave., Kansas City, Kans., ALR
SPALDING, J. A.....627 Congress St., Portland, Me., Op.A
SPENCER, F. R.....Physicians Bldg., Boulder, Colo., OALR
SPENCER, W.,
1830 S. Rittenhouse Sq., Philadelphia, Pa., OALR
*SPENGLER, J. A.....423 Main St., Geneva, N. Y., OALR
SPIECE, W. K.....Heyworth Bldg., Chicago, Ill., OALR
SPITZE, E. C.....Murphy Bldg., East St. Louis, Ill., Op.
STAHLMAN, F. C.....318 Fifth St., Charleroi, Pa., OALR
*STANBERRY, H.....Doctors Bldg., Cincinnati, O., Op.AR
STANFORD, J. B.....Exchange Bldg., Memphis, Tenn., OALR
STARR, E. G.....523 Delaware Ave., Buffalo, N. Y., Op.
STAUFFER, F...Deseret Bk. Bldg., Salt Lake City, Utah, OALR
STEGMAN, L. B.....Sanitarium, Battle Creek, Mich., Op.

STEIM, J. M.,
First Natl. Bank Bldg., New Kensington, Pa., Op.
STEIN, JULES C...22 E. Washington St., Chicago, Ill., Op.
STEIN, O. J.....30 N. Michigan Blvd., Chicago, Ill., ALR
*STEINFELD, A. L.....234 Michigan St., Toledo, O., OALR
STELLE, HARRY L.,
207 Commerce Bldg., Pittsburg, Kan., OALR
*STEVENS, H. B.,
522 Commonwealth Ave., Boston, Mass., Op.
STEVENSON, R.....11½ E. 8th St., Cincinnati, O., ALR
STEVENSON, W. D.....165 E. Market St., Akron, O., OALR
STEVENSON, W. D.....Majestic Bldg., Quincy, Ill., OALR
STEWART, C. S.....106 N. Main St., Auburn, Ind., OALR
*STEWART, WALTER E.,
Tribune Bldg., Terre Haute, Ind., OALR
*STICKNEY, OTIS D.,
92 Pacific Ave., Atlantic City, N. J., ALR
STIEREN, E....626 Union Trust Bldg., Pittsburgh, Pa., Op.
STILES, C. M.....1831 Chestnut St., Philadelphia, Pa., Op.
STILLSON, H.....Seaboard Bldg., Seattle, Wash., OALR
STIMSON, GEORGE W.,
Jenkins Arcade Bldg., Pittsburgh, Pa., ALR
STOCKLER, J. A.....332 North Fifth St., Reading, Pa., OALR
STOTTER, ARTHUR L...1148 Euclid Ave., Cleveland, O., OALR
STOTTER, J.....1148 Euclid Ave., Cleveland, O., OALR
*STOUT, PHILIP S.,
Medical Arts Bldg., Philadelphia, Pa., ALR
*STRADER, G. L.....408 Hynds Bldg., Cheyenne, Wyo., OALR
STRICKER, L.....The Groton, Cincinnati, O., Op.
STRICKLER, D. A.....615 Empire Bldg., Denver, Colo., OALR
STRONG, J. C.,
American Natl. Bank Bldg., Leadville, Colo., OALR
STROUSE, FRED. M...1301 Spruce St., Philadelphia, Pa., ALR
STROUT, E. S....Donaldson Bldg., Minneapolis, Minn., OALR
STROUT, G. ELMER,
Donaldson Bldg., Minneapolis, Minn., OALR
STUEBER, F. G.....Main and North Sts., Lima, O., OALR
STUEBER, P. J.....209 W. North St., Lima, O., OALR
STUCKY, J. A.,
Fayette Natl. Bank Bldg., Lexington, Ky., OALR
STUCKY, W. S.....2d and Upper Sts., Lexington, Ky., OALR
STURGIS, L. F.,
128 W. 87th St., New York City, N. Y., ALR
STURM, S. A...Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
SUFFA, G. A.....Gulford Hall, Boston, Mass., Op.
*SUKER, G. F.....25 E. Washington St., Chicago, Ill., Op.
*SULLIVAN, JOHN J. JR.,
Traders Bank Bldg., Scranton, Pa., ALR
SULZER, G. A.....200 E. State St., Columbus, O., OALR
*SULZMAN, F. M.....1831 Fifth Ave., Troy, N. Y., OALR

SWAN, C. J.....The Gables Bldg., Evanston, Ill., OALR
SWEET, W. M.....1205 Spruce St., Philadelphia, Pa., Op.
SWETNAM, C. R. K...Masonic Temple, Prescott, Ariz., ALR

TANGEMAN, H. F.,
607 Union Central Bldg., Cincinnati, O., ALR
*TAQUINIO, GEORGE J.,
Maison Blanche Bldg., New Orleans, La., ALR
TARUN, W.....605 Park Ave., Baltimore, Md., Op.A
TAYLOR, CHAS. B.....Ennis Bldg., Ottumwa, Ia., ALR
TAYLOR, L. E. Marshall Field Annex Bldg., Chicago, Ill., ALR
*TAYLOR, LEWIS H.,
83 S. Franklin St., Wilkes-Barre, Pa., OALR
*TAYLOR, M. C...415 Warrington Ave., Pittsburgh, Pa., Op.
*THIGPEN, C. A.,
First Natl. Bank Bldg., Montgomery, Ala., OALR
THOMAS, CHAS. D.,
Central Natl. Bank Bldg., Peoria, Ill., OALR
THOMAS, FRANCES W.....74 S. 5th St., Columbus, O., OALR
THOMAS, H. G.....Dalziel Bldg., Oakland, Cal., OALR
THOMASON, H. E.....Rialto Bldg., Kansas City, Mo., OALR
THOMASSON, W. J.....942 York St., Newport, Ky., OALR
THOMPSON, E. H.,
Vindonissa Bldg., 19 W. 7th St., Cincinnati, O., OALR
*THOMPSON, G. H.,
18 Ashland St., North Adams, Mass., OALR
THOMPSON, H. M.,
First Natl. Bank Bldg., Pueblo, Colo., Op.
THOMPSON, P. H.,
308 Commonwealth Ave., Boston, Mass., Op.
THOMSON, J. J.....3 Park Ave., Mt. Vernon, N. Y., OALR
TILDERQUIST, D. L.....7 E. Superior St., Duluth, Minn., OALR
TIMBERMAN, A.....City Bk. Bldg., Columbus, O., OALR
*TINDOLPH, L. W.....1st Natl. Bk. Bldg., Olean, N. Y., OALR
*TINGLEY, L. P...9 Massachusetts Ave., Boston, Mass., Op.
TITSWORTH, GUY.....111 W. 4th St., Sedalia, Mo., Op.
TIVNEN, R. J.....104 S. Michigan Blvd., Chicago, Ill., OALR
*TOBEY, G. L...270 Commonwealth Ave., Boston, Mass., ALR
TODD, G. C..307 Westinghouse Bldg., Pittsburgh, Pa., OALR
TODD, H. COULTER,
Colcord Bldg., Oklahoma City, Okla., OALR
TOMASSENE, RAYMOND A.,
Wheeling Steel Corp. Bldg., Wheeling, W. Va., OALR
TOMLIN, W. S.,
Hume-Mansur Bldg., Indianapolis, Ind., ALR
TOMS, S. W. S.....Nyack-on-Hudson, N. Y., ALR
TOOKER, C. W.....Carleton Bldg., St. Louis, Mo., Op.
TOWNSEND, JOHN F.,
85 Wentworth, Cor. St. Phillips St., Charleston, S. C., OALR
TRIBLE, GEORGE B.,
1026 16th St., N. W., Washington, D. C., OALR

TRIMBLE, CLARENCE S.,
705 Commercial St., Emporia, Kan., OALR
TRIPP, IRA C.....436 Rose Bldg., Cleveland, O., OALR
TRIPP, LEROY R.,
307 Security Bldg., Sioux City, Iowa, OALR
*TUCKER, GABRIEL F.....335 Trevor Lane, Cynwyd, Pa., ALR
TUCKER, JAMES C.....Beatrice, Neb., OALR
*TUCKERMAN, W. C.....Osborn Bldg., Cleveland, O., Op.
*TUCKERMAN, W. H.....Osborn Bldg., Cleveland, O., OALR
TUREMAN, HERBERT G.,
1100 Rialto Bldg., Kansas City, Mo., OALR
TURNBULL, F. M.,
504 Providence Bldg., Duluth, Minn., ALR
*TURNER, H. H.,
Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
TURNER, O. W....Augusta Trust Bldg., Augusta, Me., OALR
TWITTY, W. C.,
Peoples Natl. Bank Bldg., Rock Hills, S. C., OALR
TYDINGS, O.....31 N. State St., Chicago, Ill., OALR

UPHAM, H. F.....305 Third Ave., Asbury Park, N. J., OALR
*UREN, CLAUDE THOMAS,
325 Omaha Natl. Bank Bldg., Omaha, Neb., OALR
URNER, M. H.,
Union Central Life Bldg., Cincinnati, O., OALR

VAIL, D. T.....24 E. 8th St., Cincinnati, O., Op.
VAIL, H. H.....24 E. 8th St., Cincinnati, O., ALR
VAN KIRK, FRANK J.,
Bell. Natl. Bank Bldg., Bellingham, Wash., OALR
VANKIRK, V. E..Union Arcade Bldg., Pittsburgh, Pa., Op.
VAN POOLE, GIDEON McD.,
Young Bldg., Honolulu, Hawaii, OALR
VAUGHAN, G. E.....Franklin St., Southside, Tenn., OALR
VAUGHN, EDGAR H.,
113½ W. Ferguson St., Tyler, Tex., OALR
VEASEY, C. A.....Paulsen Bldg., Spokane, Wash., OALR
VINSONHALER, F...Urquhart Bldg., Little Rock, Ark., OALR
*VOIGHT, C. B.....1702 Broadway, Mattoon, Ill., OALR
VON COLDITZ, G. F.,
25 E. Washington St., Chicago, Ill., OALR
VOORHEES, I. W.,
14 Central Park, W., New York City, N. Y., ALR

WAGERS, ARTHUR J.,
The Lenox, 13th and Spruce Sts., Philadelphia, Pa., ALR
WAGNER, C. B.....31 North State St., Chicago, Ill., OALR
WALES, E. DEWOLF,
1236 N. Penn St., Indianapolis, Ind., ALR

WALKER, CHAS. C.,
Woodward at Warren, Detroit, Mich., Op.
WALKER, C. E.....Jacobson Bldg., Denver, Colo., Op.
WALKER, H. S....77 E. Washington St., Chicago, Ill., OALR
WALLACE, F. E....517 Thatcher Bldg., Pueblo, Colo., OALR
WALSH, JOSEPH M.....Rapid City, S. D., OALR
WALTER, WILL.....1414 Chicago Ave., Evanston, Ill., OALR
WATSON, H. D.....151 Front St., Binghampton, N. Y., OALR
WATSON, JAMES A.,
500 Phys. and Surg. Bldg., Minneapolis, Minn., OALR
*WATSON, R. SEYMOUR,
120 N. Michigan Ave., Saginaw, Mich., OALR
WAUGH, J. M.,
Cleveland Clinic, Euclid Ave. at 93rd St., Cleveland, O., LR
WEAVER, T. W.....201 N. Main St., Wichita, Kan., Op.
WEED, H. M.....196 Lincoln Ave., Buffalo, N. Y., Op.
WEEKS, JOHN E.,
20 E. 53rd St., New York City, N. Y., Op.
WEEKS, W. W.....20 E. 53rd St., New York, N. Y., Op.
WEIH, ELMER PAUL..605 Wilson Bldg., Clinton, Iowa, OALR
*WEIL, A. L.,
717 Maison Blanche Annex, New Orleans, La., ALR
WEIMER, E. S.....332 Lehigh Ave., Pittsburgh, Pa., Op.
WEISSER, E. A.....Empire Bldg., Pittsburgh, Pa., Op.
WELCH, R. S. G.,
League Island Nav. Hosp., Philadelphia, Pa., OALR
*WELLS, D. W.....Hotel Westminster, Boston, Mass., Op.
WELLS, G. S.,
San Marcos Bldg., Santa Barbara, Cal., ALR
WELLS, W. A..The Rochambeau, Washington, D. C., OALR
WELSH, D. E.,
Powers Theater Bldg., Grand Rapids, Mich., OALR
WELTY, C. F.....Shreve Bldg., San Francisco, Cal., ALR
WENCKE, C. G.,
16 Wentworth Court, Battle Creek, Mich., OALR
WENDEL, JACOB S.,
David Whitney Bldg., Detroit, Mich., ALR
WESTCOTT, C. D.....22 E. Washington, Chicago, Ill., Op.
WESTLAKE, S. V.....Humboldt Bldg., St. Louis, Mo., ALR
WHALEY, E. M..1430 Blanding Bldg., Columbia, S. C., OALR
WHEELER, ERNEST CALTRON,
Tacoma Clinic, Tacoma, Wash., OALR
*WHEELER, JOHN M.,
30 W. 59th St., New York City, N. Y., Op.
WHEELER, MERRITT W..Lowry Bldg., St. Paul, Minn., OALR
*WHERRY, W. P.,
620 Bankers Reserve Bldg., Omaha, Neb., OALR
WHISNANT, A. M.....Realty Bldg., Charlotte, N. C., OALR
WHITAKER, J..Hume-Mansur Bldg., Indianapolis, Ind., OALR
*WHITE, C. P.....2327 W. 18th St., Wilmington, Del., OALR
*WHITE, E. H.....589 Dorchester St., Montreal, Can., ALR

*White, L. E..390 Commonwealth Ave., Boston, Mass., ALR
Wible, E. E...Diamond Bank Bldg., Pittsburgh, Pa., Op.
Wiener, A.....550 Park Ave., New York City, N. Y., OALR
Wiener, M.....Carleton Bldg., St. Louis, Mo., Op.
Wilbur, E. P.,
Kalamazoo Natl. Bk. Bldg., Kalamazoo, Mich., OALR
Wilder, W. H.....122 S. Michigan Blvd., Chicago, Ill., Op.
*Willard, L. M.....520, 3rd St., Wausau, Wis., OALR
Williams, Horace J.,
5908 Green St., Germantown, Philadelphia, Pa., ALR
Williams, J. P.....Funke Bldg., Lincoln, Neb., OALR
Williams, Thos. J..30 N. Michigan Blvd., Chicago, Ill., OALR
Williams, W. C.....Jefferson Bldg., Peoria, Ill., OALR
*Williams, Walter H.....Middletown, O., OALR
Wilmer, Francis M....Castell Bldg., Winfield, Kan., OALR
Wilson, R. C.....208 E. State St., Ithaca, N. Y., OALR
*Winter, G. E.....44 Sun Bldg., Jackson, Mich., OALR
Winter, John Arthur..Fidelity Bldg., Duluth, Minn., OALR
*Wise, Ralph C.....News Bldg., Mansfield, O., OALR
Wold, Karl C...7th and Wacouta Sts., St. Paul, Minn., OALR
Wolfe, A. C.....350 E. State St., Columbus, O., ALR
*Wolfe, C. T.....Francis Bldg., Louisville, Ky., OALR
*Wolfe, Otis R..Masonic Temple, Marshalltown, Iowa, OALR
Wolfenstein, Leo.....746 Rose Bldg., Cleveland, O., Op.
Wood, C. A.....7 W. Madison St., Chicago, Ill., Op.
Wood, D..The Donaldson Bldg., Minneapolis, Minn., OALR
Wood, Hilliard..Independent Bldg., Nashville, Tenn., OALR
Woodruff, F. E...Metropolitan Bldg., St. Louis, Mo., Op.
Woodruff, H. W.....Woodruff Bldg., Joliet, Ill., OALR
Woodruff, T. A.....Plant Bldg., New London, Conn., Op.
*Woods, Hiram.....842 Park Ave., Baltimore, Md., Op.A
Woods, R. H.....Neustadt Bldg., La Salle, Ill., OALR
Woodson, J. M.,
City National Bank Bldg., Temple, Tex., OALR
*Woodward, J. M.....Richards Blk., Lincoln, Neb.
Wormley, H. R.....410 Brown Bldg., Rockford, Ill., OALR
Wright, Chas. D.,
1111 Metropolitan Bk. Bldg., Minneapolis, Minn., OA
Wright, J. B..Citizens Natl. Bk. Bldg., Raleigh, N. C., OALR
Wright, J. W..Central Natl. Bk. Bldg., Columbus, O., Op.
*Wright, J. W. M.,
Hume-Mansur Bldg., Indianapolis, Ind., ALR
Wright, K. L.....247 W. 8th St., Erie, Pa., ALR
*Wurtz, W. J. M..2808 Delaware Ave., Kenmore, N. Y., ALR
Würdemann, H. V.....Cobb Bldg., Seattle, Wash., OALR
Wylie, C. B.....293 High St., Morgantown, W. Va., OALR

Yankauer, S..616 Madison Ave., New York City, N. Y., ALR
Yazel, Herman E.,
510-511 Grand Ave. Temple, Kansas City, Mo., OALR

YOUNG, H. B.....204 Jefferson St., Burlington, Ia., OALR
YOUNG, H. H.....Morrison Bldg., Charleston, W. Va., OALR

*ZEHDER, C. A.....180 Fairmount Ave., Newark, N. J., OALR
ZEMER, S.....Security Mutual Bldg., Lincoln, Neb., OALR
ZENTMAYER, WILLIAM,
1506 Spruce St., Philadelphia, Pa., Op.
ZIEGLER, S. L.....1625 Walnut St., Philadelphia, Pa., Op.
ZINKHAN, ARTHUR M.,
1025 Fiftenth St., N. W., Washington, D. C., ALR

GEOGRAPHIC LIST

ALABAMA

BIRMINGHAM

HARRISON, W. G. ..... Empire Bldg., OALR
RUCKER, EDMOND W. J. ..... Woodward Bldg., ALR

MOBILE

MAUMENEE, A. E. ..... 156 St. Francis St., OALR

MONTGOMERY

THIGPEN, C. A. ..... First National Bank Bldg., OALR

SELMA

KIRKPATRICK, SAMUEL ..... Lauerdale Ave., OALR

TUSCALOOSA

SEARCY, H. B. ..... 807 Greensboro Ave., OALR

ARIZONA

PHOENIX

MARTIN, ANCIL ..... 207 Goodrich Bldg., Op.A

PRESCOTT

SWETNAM, C. R. K. ..... Masonic Temple, ALR

ARKANSAS

EUREKA SPRINGS

HUNTINGTON, ROBT. H. ..... OALR

FORT SMITH

BUCKLEY, J. H. ..... 1st Natl. Bank Bldg., OALR
DORENTO, DRED R. ..... Merchants National Bank Bldg., OALR
MOULTON, EVERETT C. ..... Merchants National Bank Bldg., OALR
MOULTON, HERBERT ..... Merchants National Bank Bldg., OALR

HOT SPRINGS

KING, O. H. ..... Dugan-Stuart Bldg., OALR
ROWLAND, J. F. ..... Thompson Bldg., OALR
SHERMAN, MAJ. A. E. ..... Army & Navy Gen. Hosp., OALR
SHORT, Z. N. ..... Dugan-Stuart Bldg., OALR

LITTLE ROCK

CALDWELL, ROBERT ..... State National Bank Bldg., OALR
VINSONHALER, FRANK ..... Urquhart Bldg., OALR

PINE BLUFF

CRUMP, J. F.....Citizens' Bank Bldg., OALR
SCALES, J. W.....202½ Main St., OALR

ROGERS

KOOBS, H. J. G.....4th and Walnut Sts., OALR

CALIFORNIA

HOLLYWOOD

KERRICK, STANLEY E.....Security Bldg., OALR

LONG BEACH

MEANS, C. S.....Pacific-Southwest Bldg., OALR

LOS ANGELES

BELLIN, JOSEPH.....1026 W. 6th St., OALR
BOURBON, OLIVER P...222 Westlake Professional Bldg., OALR
BROWN, J. MACKENZIE.....1136 W. 6th St., ALR
DETLING, F.....Investment Bldg., OALR
DUDLEY, WM. H.....Brockman Bldg., OALR
FISHER, CARL.....1028 Pacific Mut. Bldg., Op.A
GOSNEY, C. W.....6075 Franklin Ave., OALR
IRVINE, A. R.....709 Westlake Professional Bldg., Op.
KIEFER, HUGO A.....Brockman Bldg.,OALR
KRESS, GEO. H.....Bradbury Bldg., OALR
LEFFLER, ANNA B.....W. P. Story Bldg., OALR
MANSUR, D. W.....Brockman Bldg., Op.

OAKLAND

O'CONNOR, R. P.....1206 Broadway, Op.
THOMAS, H. C.....Dalziel Bldg., OALR

PASADENA

HENNINGER, L. L.....Citizens Bank Bldg., OALR
ROBERTS, W. H.....461 E. Colorado St., OALR

SACRAMENTO

BRIGGS, WM. E.....Box 166, OALR

SAN DIEGO

BURTON, F. A.....404 Watts Bldg., ALR

SAN FRANCISCO

BARKAN, HANS.....Physicians Bldg. Op.
BLAKE, WM. FORD.....516 Sutter St., Op.
BROWN, H. A.....719 St. Paul Bldg., Op.
DOLMAN, P.....Flood Bldg., Op.
GRAHAM, H. B.....210 Post St., ALR
GREEN, AARON S.....853 Hyde St., Op.
GREEN, LOUIS D.....853 Hyde St., Op.
ISRAEL, J. P.....U. S. Veterans Bureau, OALR
LEWITT, F. C.....135 Stockton St., OAL
McNAUGHT, H.....Butler Bldg., ALR

deRIVER, JOSEPH.....U. S. Veterans Bureau, OALR
SELFRIDGE, G. L.....Schroth Bldg., ALR
SEWALL, E. C.....Lane Hospital, OALR
WELTY, C. F.....Shreeve Bldg., ALR

SANTA BARBARA

JEAN, G. W.....San Marcos Bldg., Op.
RYAN, LAWRENCE R.....San Marcos Bldg., Op.A
WELLS, GEO. S.....San Marcos Bldg., ALR

COLORADO

ALAMOSA

HERRIMAN, L. L.....Physicians Bldg., OALR

BOULDER

FISCHER, VALENTINE B.....Mercantile Bank Bldg., OALR
LaRUE, C. L.....17 Physicians Bldg., OALR
SPENCER, F. R.....Physicians Bldg., OALR

COLORADO SPRINGS

DENNIS, FRANK L.....Burns Bldg., ALR
MAGRUDER, A. C.....Burns Bldg., OALR
MULLIN, W. V.....Burns Bldg., ALR
NEEPER, EDW. R.....Exchange Natl. Bank Bldg., Op.
PATTERSON, JAS. A.....Burns Bldg., OALR

DELTA

SMITH, H. A.....6540 Main St., OALR

DENVER

ALLEN, JOS. H.....Majestic Bldg., ALR
BANE, WILLIAM.....Metropolitan Bldg., OALR
BANE, W. C.....Metropolitan Bldg., OALR
BAUM, H. L.....Metropolitan Bldg., ALR
BLACK, MELVILLE.....Metropolitan Bldg., Op.
BOYD, E. T.....Majestic Bldg., OALR
BROWN, MORROW D.....Metropolitan Bldg., ALR
CARMODY, T. E.....Metropolitan Bldg., ALR
COLLINS, E. W.....Majestic Bldg., ALR
COOPER, C. E.....Metropolitan Bldg., ALR
COOVER, D. H.....Metropolitan Bldg., Op.
CRISP, WM. H.....Metropolitan Bldg., Op.
FINNOFF, WM. C.....Imperial Bldg., Op.A
FOLEY, J. W.....Majestic Bldg., OALR
FOSTER, J. M.....Metropolitan Bldg., OALR
GALE, M. J.....Imperial Bldg., Op.
GALLAHER, T. J.....California Bldg., ALR
HALSTED, FRED S.....Metropolitan Bldg., OALR
HESS, WM. L.....1554 California St., OALR
JACKSON, E.....Imperial Bldg., Op.
LEVY, ROBERT.....Metropolitan Bldg., ALR

LIBBY, GEO. F.....Majestic Bldg., Op.
MCCAW, J. A.....Majestic Bldg., Op.
MCKEOWN, ELMER E.....Metropolitan Bldg., OALR
MONAGHAN, DANIEL G.....Majestic Bldg., Op.
SEDWICK, WM. A.....Imperial Bldg., OALR
SHIELDS, J. MELVILLE.....Metropolitan Bldg., Op.
STRICKLER, D. A.....Empire Bldg., OALR
WALKER, C. E.....Jacobson Bldg., Op.

GRAND JUNCTION

CARY, G. C..... OALR

GREELEY

LEHAN, JAS. W.....Park Pl. Bldg., OALR
RINGLE, CHAS. A.....Coronado Bldg., ALR

LEADVILLE

STRONG, JAS. C.....Amer. National Bank Bldg., OALR

PUEBLO

LASSEN, FRITZ.....First National Bank Bldg., ALR
THOMPSON, H. M.....First National Bank Bldg., Op.
WALLACE, FRANKLIN E.....Thatcher Bldg., OALR

TRINIDAD

RICHIE, LEE T.....McCormick Bldg., OALR

CONNECTICUT

BRIDGEPORT

SMITH, DORLAND.....834 Myrtle Ave., Op.A

DANIELSON

BURROUGH, G. M.....5 Broad St., Op.

HARTFORD

HOGAN, WALTER L.....750 Main St., OALR
SMITH, E. TERRY.....Connecticut Mutual Bldg., Op.

NEW LONDON

WOODRUFF, T. A.....Plant Bldg., Op.

DELAWARE

WILMINGTON

ELLEGOOD, J. A.....Equitable Bldg., OALR
WHITE, C. P.....2327 W. 18th St., OALR

DISTRICT OF COLUMBIA

WASHINGTON

DABNEY, VIRGINIUS.....1633 Connecticut Ave., ALR
DAVIS, W. T.....927 Farragut Sq., Op.
DEWEY, C. H.....Walter Reed Gen. Hospital

FLYNN, JAMES A.....1616 Eye St., ALR
GILL, G. BACHE.....1624 Eye St., ALR
HENNING, CARL.....The Rochambeau, Op.
LAMB, ROBERT S.....Stoneleigh Court, Op.
MOORE, MEADE.....The Rochambeau, ALR
MUNCASTER, S. B.....921, 15 A St., Op.A
NEWELL, WM. S.....1209 Vermont Ave.
PARRISH, MAJ. ROBERT E.,
M. C., U. S. A., Walter Reed Gen. Hospital, ALR
RICHARDSON, CHAS. W.....1317 Connecticut Ave., ALR
RICHARDSON, JAS. J.....1509 Avenue of Presidents, ALR
SCHLANZER, A. E....Walter Reed U. S. A. Gen. Hosp., OALR
SHUTE, D. K.....1719 De Sales St., Op.
TRIBLE, GEORGE B.....1026, 16th St., N. W., OALR
WELLS, W. A.....The Rochambeau, OALR
ZINKHAN, A. M.....1025 Fifteenth St., N. W., ALR

FLORIDA

JACKSONVILLE

HEGGIE, N. H.....Buckman Bldg., OALR

KEY WEST

DEBOE, M. P.....621 Eaton St., OALR

MIAMI

HODSON, B. F.....25 N. E. First Ave., OALR

OLANDA

INGRAM, L. C. M.....Clinic Bldg., OALR

PENSACOLA

LISCHKOFF, MOZART.....Blount Bldg., OALR

TAMPA

FORBES, S. B.....Citizens' Bank Bldg., OALR

WEST PALM BEACH

MILLER, ALICE R..... OALR

GEORGIA

ATLANTA

CALHOUN, F. P.....Candler Bldg., OALR
CLAY, G. E.....Candler Bldg., OALR
FORT, A. G.....Candler Bldg., OALR
LOCKEY, H. M.....Doctors Bldg., OALR

AUGUSTA

OERTEL, T. E.....638 Green St., OALR

ROME

SMITH, GEO. B.....Broad St. and Fourth Ave., OALR

SAVANNAH

MARTIN, H. H.....247 Bull St., OALR

VALDOSTA

SMITH, J. M.....134½ N. Patterson St., OALR

HAWAII

HONOLULU

MORGAN, J. A.....Young Bldg., OALR
VAN POOLE, GIDEON McD.....Young Bldg., OALR

IDAHO

BOISE CITY

MAXEY, E. E.....Idaho Bldg., OALR

NAMPA

SHAWHAN, G. E.....OALR

ILLINOIS

ALTON

JONES, F. W.....Times Bldg., OALR
MIDDLETON, H. E.....Times Bldg., OALR

AURORA

DARMER, GEORGE A.....401 Terminal Bldg., OALR

BELLEVILLE

AUTEN, FRANK E.....First National Bank Bldg., OALR

BLOOMINGTON

FOX, RALPH D.....Unity Bldg., OALR
GAILEY, WILLIAM W.....615 Griesheim Bldg., OALR

CHICAGO

ALLPORT, FRANK.....7 W. Madison St., OALR
ANDREWS, A. H.....30 N. Michigan Blvd., OALR
ANDREWS, B. F.....25 E. Washington St., OALR
AVERY, F. T.....357 W. 63rd St., Op.LR
BALLENGER, H. C.....25 E. Washington St., ALR
BARNES, L. E.....6860 S. Halsted St., OALR
BECK, JOS. C.....2551 N. Clark St., OALR
BEEM, IONE F.....31 N. State St., OALR
BERGERON, J. Z.....104 S. Michigan Blvd., ALR
BRAWLEY, F. E.....30 N. Michigan Blvd., ALR
BUCK, ROBERT H.....15 E. Washington St., OALR
CARNCROSS, HELEN.....22 E. Washington St., Op.
CAVANAUGH, J. A.....7 W. Madison St., ALR
CROSSLEY, E. R.....29 E. Madison St., OALR
CUSHMAN, BEULAH.....25 E. Washinton St., Op.
DAVIDSON, HERMAN P.....22 E. Washington St., Op.
DENNIS, GEO. J.....25 E. Washington St., ALR

EVENSEN, H. O.....25 E. Washington St., OALR
FAITH, THOS.....31 N. State St., OALR
FARRELL, P. J.....25 E. Washington St., OALR
FELLOWS, G. G.....30 N. Michigan Blvd., OALR
FISHER, WM. A.....31 N. State St., OALR
FISKE, GEO. F.....25 E. Washington St., OALR
FRANK, IRA.....104 S. Michigan Blvd., ALR
GARRAGHAN, E. F.....25 E. Washington St., OALR
GRADLE, HARRY S.....110 N. Wabash Ave., Op.
GRIMM, U. J.....30 N. Michigan Blvd., OALR
HALL, ALICE K.....25 E. Washington St., ALR
HAWLEY, C. W.....30 N. Michigan Blvd., OALR
HAYDEN, A. A.....25 E. Washington St., OALR
JORDAN, G. T.....30 N. Michigan Blvd., OALR
KAHN, HARRY.....104 S. Michigan Blvd., ALR
KAHN, MYRON E.....29 E. Madison St., ALR
KLEENE, FREDERICK.....1445 Wicker Park Ave., OALR
KRAFT, OSCAR H.....25 E. Washington St., Op.
LOEB, CLARENCE .....25 E. Washington St., Op.
LORING, J. BROWN.....25 E. Washington St., OALR
LYONS, HORACE R.....30 N. Michigan Blvd., ALR
MARQUIS, GEO. P.....30 N. Michigan Blvd., ALR
MCBEAN, G. M.....22 E. Washington St., OALR
MCGINNIS, EDWIN.....104 S. Michigan Blvd., ALR
MUNDT, G. H.....25 E. Washington St., OALR
MURRAY, ALFRED N.....4654 Sheridan Rd., OALR
NANCE, WILLIS O.....30 N. Michigan Blvd., OALR
NORCROSS, EDWARD P.....30 N. Michigan Blvd., ALR
NOVAK, F. JR.....30 N. Michigan Blvd., ALR
ORCUT, D. C.....7 W. Madison St., Op.A
PEARLMAN, SAMUEL J.....29 E. Madison St., ALR
POLLOCK, H. L.....2551 N. Clark St., ALR
REMMENS, NILS.....2954 Logan Blvd., Op.
ROBERTSON, CHAS. M.....30 N. Michigan Blvd., OALR
SALINGER, S.....25 E. Washington St., ALR
SONNENSCHEIN, ROBERT .....Heyworth Bldg., ALR
SPIECE, W. KOENIG.....29 E. Madison St., OALR
STEIN, JULES C.....22 E. Washington St., Op.
STEIN, O. J.....30 N. Michigan Blvd., ALR
SUKER, G. F.....25 E. Washington St., Op.
TAYLOR, LILLIAN E.....Marshall Field Annex Bldg., ALR
TIVNEN, R. J.....104 S. Michigan Blvd., OALR
TYDINGS, OLIVER.....31 N. State St., OALR
VON COLDITZ, G. F.....25 E. Washington St., OALR
WAGNER, CARL B.....31 N. State St., OALR
WALKER, HERBERT S.....77 E. Washington St., OALR
WESTCOTT, C. D.....22 E. Washington St., Op.
WILDER, W. H.....122 S. Michigan Blvd., Op.
WILLIAM, T. J.....30 N. Michigan Blvd., OALR
WOOD, CASEY A.....7 W. Madison St., Op.

CHICAGO HEIGHTS

GORRELL, T. J. H.....16th and Vincennes Aves., OALR

EAST ST. LOUIS

SPITZE, E. C.....Murphy Bldg., Op.

EVANSTON

BOOT, G. W.....603 Main St., OALR
DODD, O.....708 Church St., Op.A
SWAN, C. J.....The Gables Bldg., OALR
WALTER, WILL.....1414 Chicago Ave., OALR

FREEPORT

BUCHANAN, JAMES N.....Tarbox Bldg., OALR
CLARK, J. S.....67 Stephenson St., OALR
RIDEOUT, W. J.....90 Stephenson St., OALR

GALESBURG

MATHENY, R. C.....306 E. Main St., OALR

HOOPESTON

EAREL, A. M.....507 Wildon Blk., OALR

JACKSONVILLE

ADAMS, A. L.....323 W. State St., OALR

JOLIET

WOODRUFF, H. W.....Woodruff Bldg., OALR

KANKAKEE

GEIGER, C. W.....Cobb Bldg., OALR

LA SALLE

WOODS, R. H.....Neustadt Bldg., OALR

MACOMB

DUNTLEY, G. S.....220 E. Carroll St., OALR

MATTOON

VOIGHT, C. B.....1702 Broadway, OALR

MOLINE

FLATLEY, T. J.....Reliance Bldg., OALR

PEKIN

GLASFORD, SAMUEL T.....OALR

PEORIA

BLICKENSTAFF, A. J.....804 Peoria Life Bldg., OALR
BROBST, C. H.....Central Bank Bldg., OALR
DUANE, J. F.....Jefferson Bldg., OALR
FLOYD, T. W.....Lehman Bldg., OALR
THOMAS, C. D.....Central National Bank Bldg., OALR
WILLIAMS, W. C.....Jefferson Bldg., OALR

PONTIAC

MIDDLETON, A. B.....201 Sterry Blk., OALR

QUINCY

MERCER, RAY.....Stein Bldg., OALR
STEVENSON, W. D.....Majestic Bldg., OALR

ROCKFORD

FRINGER, W. R.....William Brown Bldg., Op.
KEITH, D. M.....414 N. Main St., ALR

SPRINGFIELD

HAGLER, A. L.....Hagler Bldg., OALR
HAGLER, E. E.....Hagler Bldg., OALR

STERLING

BRODICK, FRANK W.....511 Lawrence Bldg., OALR

WAUKEGAN

GAVIN, E. F.....1 S. Sheridan Rd., OALR

INDIANA

AUBURN

STEWART, C. C.....106 N. Main St., OALR

ELKHART

PORTER, J. B.....415 S. 2nd St., OALR

EVANSVILLE

BROSE, L. D.....501 Upper First St., OALR
KNAPP, BLEEKER.....Intermediate Life Bldg., OALR
RAVDIN, B. D.....223 Main St., OALR
RAVDIN, MARCUS.....223 Main St., OALR

FORT WAYNE

BARTHOLOMEY, A. C.....122 E. Washington St., OALR
BULSON, A. E. JR.....406 W. Berry St., OALR
MCBRIDE, W. O.....218 Washington Blvd., OALR

HAMMOND

LLOYD, A. W.....Hammond Bldg., OALR
SHANKLIN, E. M.....575 Hohman St., OALR

HARTFORD CITY

HOLLIS, W. A.....214 N. High St., OALR

INDIANAPOLIS

ADAMS, DANIEL S.....Hume-Mansur Bldg., ALR
BARNHILL, J. F.....Pennway Bldg., ALR
BERRY, D. F.....Hume-Mansur Bldg., ALR
CARMACK, J. W.....37 Willoughby St., AR
CLEVENGER, W. F.....Newton-Claypool Bldg., ALR
CRAFT, KENNETH L.....Hume-Mansur Bldg., OALR

HOOD, T. C.....Hume-Mansur Bldg., Op.
HUGHES, W. F.....Hume-Mansur Bldg., Op.
KEARBY, D. O.....American Central Life Bldg., ALR
LARKIN, B. J.....Hume-Mansur Bldg., Op.
MCCASKEY, B. J.....American Central Life Bldg., ALR
MOLT, WM. F.....Newton-Claypool Bldg., ALR
MORRISON, FRANK.....Willoughby Bldg., Op.
OVERMAN, F. V.....Hume-Mansur Bldg., ALR
PAGE, LAFAYETTE.....Hume-Mansur Bldg., ALR
PFAFFLIN, C. A.....Newton-Claypool Bldg., OALR
REPASS, ROBERT E.....150 Maple Rd., ALR
ROW, CHAS. S.....Indiana Pythian Bldg., Op.
TOMLIN, W. S.....Hume-Mansur Bldg., ALR
WALES, E. DEW.....1236 N. Penn, ALR
WHITAKER, JOEL.....Hume-Mansur Bldg., OALR
WRIGHT, J. W. M.....Hume-Mansur Bldg., ALR

LAFAYETTE

KEIPER, G. F.....14 N. 6th St., OALR

MADISON

COCHRAN, R. W.....West and Third Sts., OALR

MARION

BRAUNLIN, W. H.....Marion Natl. Bank Bldg., OALR

MICHIGAN CITY

KRIEGER, G. M.....119 E. Sixth St., OALR

MUNCIE

MILLER, C. E.....315 S. Jefferson St., OALR

SOUTH BEND

BARLOW, ROY A.....461 Farmers Trust Bldg., ALR
BOONE, J. C.....Citizens Bank Bldg., OALR
BOYD-SNEE, HARRY.....716 L. M. S. Bldg., ALR
EDWARDS, S. R.....111 N. Lafayette St., OALR
HAGER, W. A.....111 N. Lafayette St., OALR
LENT, E. J.....122 N. Lafayette St., OALR
SAVERY, C. E.....126 N. Lafayette St., OALR

TERRE HAUTE

GILLUM, J. R.....221 S. 6th St., OALR
STEWART, W. E.....Tribune Bldg., OALR

UNION CITY

RUBY, F. M.....Physicians Bldg., OALR

WARSAW

HOWARD, C. N.....Warsaw Clinic., OALR

IOWA

BURLINGTON

LaForce, E. F.....Tama Bldg., OALR
Young, H. B.....204 Jefferson St., OALR

CEDAR RAPIDS

Bailey, F. W.....Security Bldg., OALR
Carroll, Frank.....Ely Block, OALR
Ivins, H. M.....408 2nd Ave., OALR

CLINTON

Weih, E. P.....Wilson Bldg., OALR

COUNCIL BLUFFS

Dean, F. W.....City National Bank Bldg., OALR
Howard, Lloyd G.....OALR
Maiden, S. D.....217 Merriam Blk., OALR

CRESTON

Childs, Hal. A.....Eagle Blk., OALR

DAVENPORT

Harkness, G. F.....2130 Main St., OALR

DES MOINES

Heard, Mary K.....Commonwealth Bldg., Op.
Linn, E. G.....Fleming Bldg., OALR
Pearson, W. W.....Bankers Trust Bldg., OALR

DUBUQUE

Langworthy, H. G.....10th and Bluff Sts., OALR

INDEPENDENCE

Agnew, F. W.....120 Third Ave., S. E., OALR

IOWA CITY

Boiler, W. F.....426 S. Dodge St., OALR
Dean, L. W.....12 S. Clinton St., OALR

KEOKUK

Lapsley, Robert M.....Masonic Temple, OALR

MARSHALLTOWN

Cobb, Edwin.....Masonic Temple, OALR
French, R. F.....Masonic Temple, OALR
Singleton, E. M.....111 E. Church St., OALR
Wolfe, O. R.....Masonic Temple, OALR

MASON CITY

Fallows, H. D.....Park Hospital Clinic, OALR
O'Brien, S. A.....First Natl. Bk. Bldg., OALR

MUSCATINE

Johnston, W. H.....Layrel Bldg., OALR

NEWTON

ENGLE, H. P.....200 Olive St., OALR

OTTUMWA

LA FORCE, BERT D.....Hofman Bldg., OALR
TAYLOR, CHAS. B.....Ennis Bldg., OALR

SIOUX CITY

DECKER, JAY C.....Francis Bldg., OALR
FRANCHERE, F. E.....4th and Nebraska Sts., OALR
GITTENS, T. R.....OALR
NAFTZGER, J. B.....Davidson Bldg., OALR
REEDER, J. E.....Davidson Bldg., OALR
ROOST, F. H.....515 Trimble Blk., OALR
SALLANDER, F. W.....Security Bk. Bldg., OALR
SECOY, F. L.....Francis Bldg., OALR
TRIPP, L. R.....307 Security Bldg., OALR

WATERLOO

JOYNT, ALBERT J.....Black Bldg., OALR
REULING, F. HAROLD.....Black Bldg., OALR
RUSSELL, RALPH E.....OALR

WAVERLY

ROBINSON, R. E.....205½ E. Bremer Ave., OALR

KANSAS

ATCHISON

BRIBACH, E. J.....Simpson Bldg., OALR

COFFEYVILLE

CHADWICK, IRA B.....Terminal Bldg., OALR

CONCORDIA

ROBERTSON, E. N.....132½ W. 6th St., OALR

ELDORADO

SHULTS, EDWIN, J. G.....Haines Bldg., OALR

EMPORIA

GRANGER, WAYNE B.....705 Commercial St., OALR
SHOEMAKER, F.....728 Union St., OALR
TRIMBLE, C. S.....705 Commercial St., OALR

FORT SCOTT

HOPPER, WILBUR L.....209 S. Main St., OALR

HIAWATHA

ROBINSON, J. M.....113 S. 6th St., OALR

HUTCHINSON

GREEVER, B. L.....State Exchange Bldg., OALR
SCALES, H. L.....Rorabaugh-Wiley Bldg., OALR
SCHRANT, J. H.....Natl. Bank Bldg., OALR

KANSAS CITY

LIDIKAY, CHAS. J.....519 Portsmouth Bldg., OALR
MCCARTNEY, OLIVER P.....314 Portsmouth Bldg., OALR
SPAKE, L. B.....522 Minnesota Ave., ALR

LEAVENWORTH

HAAS, F. J.....Masonic Bldg., OALR

OTTAWA

NEIGHBORS, C. A.....Underwood Bldg., OALR
SCOTT, J. R.....201 S. Main St., OALR

PARSONS

LANDES, G. A.....110½ S. 18th St., OALR

PITTSBURG

GRAVES, WILBUR H.....Commerce Bldg., OALR
STELLE, H. L.....Commerce Bldg., OALR

TOPEKA

ALKIRE, H. L.....614 Kansas Ave., OALR
ALLEN, G. H.....Mills Bldg., OALR
ALLEN, GEO. V.....835 Kansas Ave., OALR
ESTERLY, D. E.....Mills Bldg.
MAGEE, R. S.....Mills Bldg., Op.A

WICHITA

CHENEY, JAMES W.....Schweiter Bldg., OALR
DORSEY, J. G.....201 N. Main St., OALR
GILLETT, WILBUR G.....201 N. Main St., Op.
GSELL, J. F.....Beacon Bldg., OALR
HIGGINBOTHAM, T. L.....Solomon Clinic Bldg., OALR
HUTCHINSON, O. G.....Schweiter Bldg., OALR
MAGGARD, D. I.....Beacon Bldg., OALR
PALMER, E. M.....Beacon Bldg., OALR
SEYDELL, E. M.....201 N. Main St., ALR
WEAVER, T. W.....201 N. Main St., Op.

WINFIELD

RALLS, C. T.....First National Bank Bldg., OALR
WILMER, F. M.....Castell Bldg., OALR

KENTUCKY

COVINGTON

AUFMWASSER, H.....1302 Greenup St., OALR
BLEDSOE, R. W.....1005 Madison Ave., OALR

FULTON

COHN, SELDON.....416 Luke St., OALR

LEXINGTON

DEWEESE, CLARENCE.....154 N. Upper St., Op.
OFFUT, W. N.....Security Bldg., OALR
STUCKY, J. A.....Fayette National Bank Bldg., OALR
STUCKY, W. S.....2nd and Upper Sts., OALR

LOUISVILLE

BASS, A. L.....Francis Bldg., OALR
DABNEY, S. G.....Starks Bldg., OALR
DEAN, WALTER .....Francis Bldg., OALR
DULANEY, O.....717 Starks Bldg., OALR
HALL, G. C.....Gaulbert Bldg., OALR
HEITGER, J. D.....808 Francis Bldg., OALR
KELLY, C. W., JR.....Francis Bldg., OALR
LEDERMAN, ISAAC A.....Atherton Bldg., OALR
PFINGST, A. O.....Atherton Bldg., OALR
SHAFER, J. J.....Starks Bldg., OALR
WOLFE, CLAUDE T.....Francis Bldg., OALR

NEWPORT

THOMASSON, W. J.....942 York St., OALR

PADUCAH

REYNOLDS, H. G.....City National Bank Bldg., Op.A

LOUISIANA

NEW ORLEANS

BAHN, CHAS. A.....722 Maison Blanche Annex, Op.
BLUM, H. N.....Maison Blanche Bldg., Op.
FEINGOLD, MARCUS.....4206 St. Charles Ave., Op.
HASPEL, D. M.....1214 Maison Blanche Bldg., ALR
LANDFRIED, C. J..607 Land Title and Guarantee Bldg., ALR
LYNCH, R. C.....Maison Blanche Bldg., ALR
SMITH, V. C.....Maison Blanche Bldg., Op.
TAQUINIO, G. J.....Maison Blanche Bldg., ALR
WEIL, A. L.....717 Maison Blanche Annex Bldg., ALR

SHREVEPORT

MOISE, A. B.....Commercial Bk. Bldg., ALR
SCALES, J. L.....Commercial Bk. Bldg., OALR

MAINE

AUGUSTA

TURNER, O. W.....Augusta Trust Bldg., OALR

PORTLAND

BEACH, S. J.....704 Congress St., OALR
GILBERT, F. Y.....148 Park St., Op.AR
HASKELL, A. D.....145 High St., Op.
HOLT, E. E. JR.....723 Congress St., Op.A
SPALDING, J. A.....627 Congress St., Op.A

MARYLAND

BALTIMORE

BORDLEY, JAMES, JR.....330 N. Charles St., Op.
FRIEDENWALD, HARRY.....1029 Madison Ave., Op.A
GOLDBACH, L. J.....1012 N. Charles St., OALR
KEMLER, JOSEPH J.....1908 Eutaw Pl., OALR
LOOPER, EDWARD A.....605 Park Ave., OALR
PETERMAN, H. F.....518 N. Charles St., OALR
TARUN, WILLIAM.....605 Park Ave., Op.A
WOODS, HIRAM.....842 Park Ave., Op.A

CUMBERLAND

JONES, E. L.....First National Bank Bldg., OALR
ROBINSON, H. T.....Medical Bldg., OALR
SHARRETT, GEORGE O.....119 Bedford St., OALR

MASSACHUSETTS

BOSTON

BRYANT, A. G.....502 Beacon St., ALR
CARVILL, L. MAUD.....82 Commonwealth Ave., Op.
CHENERY, W. E.....377 Commonwealth Ave., ALR
CHENEY, F. E.....64 Commonwealth Ave., Op.
DERBY, G. S.....23 Bay State Road, Op.
FREEDMAN, L. M.....419 Boylston St., ALR
GREENWOOD, ALLEN.....82 Commonwealth Ave., Op.
HURLEY, E. D.....419 Boylston St., Op.
HURLEY, E. H.....419 Boylston St., Op.
INGLIS, H. J.....Bay State Bldg., ALR
LANCASTER, W. B.....520 Commonwealth Ave., Op.
LEMOINE, A. L.....233 Charles St., Op.
LEWIS, J. P.....543 Boylston St., ALR
MOSHER, H. P.....828 Beacon St., ALR
RICE, G. B.....293 Commonwealth Ave., ALR
RICHARDS, LYMAN G.....270 Commonwealth Ave., ALR
ROWLAND, WILLIAM D.....220 Clarendon St., Op.
SHANAHAN, T. J.....419 Boylston St., ALR
STANDISH, MYLES.....51 Hereford St., Back Bay, Op.
STEVENS, H. B.....522 Commonwealth Ave., Op.
SUFFA, G. A.....Gulford Hall, Op.
THOMPSON, P. H.....308 Commonwealth Ave., Op.
TINGLEY, LOUISA P.....9 Massachusetts Ave., Op.
TOBEY, G. L.....270 Commonwealth Ave., ALR
WELLS, D. W.....The Westminster, Op.
WHITE, L. E.....390 Commonwealth Ave., ALR

CLINTON

MONAHAN, J. A.....181 Chestnut St., OALR

FALL RIVER

RYDER, D. R.....151 Rock St., ALR

FRAMINGHAM

JESSAMAN, L. W.....182 Concord St., OALR

GARDNER

BONE, H. D.....29 Parker St., OALR

HOLYOKE

HUSSEY, E. J.....276 High St., Op.LR

LAWRENCE

CONLON, F. A.....Bay State Bldg., OALR
MERRILL, W. H.....Bay State Bldg., OALR

LYNN

JONES, E. W.....44 Atlanta St., Op.A
O'REILLY, W. F.....44 S. Common St., OALR

NEW BEDFORD

POTTER, L. F.....68 Walnut St., OALR

NORTH ADAMS

THOMPSON, G. H.....18 Ashland St., OALR

NORTHAMPTON

COLLINS, J. D.....90 Main St., ALR
DOW, F. E.....4 West St., Op.

PALMER

MOORE, G. A.....Bank Bldg., OALR

SALEM

HENNESSY, W. W.....333 Essex St., OALR

SPRINGFIELD

BYRNES, H. F.....4 Chestnut St., OALR
CARLETON, RALPH.....10 Temple St., Op.
FRELIGH, C. A.....75 State St., ALR
IRWIN, V. J.....389 Main St., OALR
IRWIN, V. J., JR.....389 Main St., OALR

TAUNTON

BAKER, H. B.....58 Main St., OALR

WESTFIELD

CLARK, F. T.....52 Broad St., OALR

WORCESTER

CAHILL, J. W.....390 Main St., OALR
CROSS, A. E.....Slater Bldg., Op.
ESTABROOK, CHARLES.....390 Main St., OALR
RICE, JOHN E.....390 Main St., OALR

MICHIGAN

ANN ARBOR

SLOCUM, GEORGE.....328 E. Huron St., Op.

BATTLE CREEK

COLVER, B. N.....182 Manchester St., OALR
FARNESWORTH, M. A.....146 Greenwood St., OALR
HAUGHEY, WILFRID.....24 W. Main St., OALR
SLEIGHT, R. D.....Post Bldg., OALR
STEGMAN, L. B.....Sanitarium, Op.
WENCKE, C. G.....16 Wentworth Ct., OALR

BAY CITY

BAKER, C. H.....Crapo Block, OALR

DETROIT

AMBERG, EMIL.....David Whitney Bldg., ALR
BEATTIE, ROBERT.....David Whitney Bldg., OALR
BENTLEY, NEIL.....1161 David Whitney Bldg., OALR
BERNSTEIN, E. J.....Professional Bldg., OALR
CAMPBELL, D. A.....David Whitney Bldg., OALR
CAMPBELL, DON M.....Peter Smith Bldg., OALR
CONNOR, RAY.....1410 Stroh Bldg., OALR
DEFNET, WM. A.....62 Adams St., W., ALR
GOUX, R. S.....545 David Whitney Bldg., OALR
HARTZ, H. J.....27 E. Adams St., ALR
HEATH, PARKER.....David Whitney Bldg., Op.
MAIRE, L. E.....Park Bldg., Op.A
MCCLELLAND, CARL C.....David Whitney Bldg., OALR
MERCER, R. E.....David Whitney Bldg., LR
MINER, S. C.....1564 Woodward Ave., ALR
MORTON, JOHN B.....90 E. Warren Ave., OALR
ODELL, ANNA.....32 Adams St., Op.AL
PARKER, WALTER R.....David Whitney Bldg., Op.
PEIRCE, HORACE W.....David Whitney Bldg., ALR
RENAUD, G. L.....Fine Arts Bldg., OALR
SANDERSON, H. H.....David Whitney Bldg., OALR
SHURLEY, B. R.....62 W. Adams St., ALR
SIMPSON, H. L.....David Whitney Bldg., ALR
WALKER, C. C.....Woodward at Warren, Op.
WENDEL, JACOB S.....David Whitney Bldg., ALR

ESCANABA

BOYCE, WILLIAM B.....1019 Ludington St., OALR

GRAND RAPIDS

DEAN, ALFRED.....304 Gilbert Blk. Bldg., Op.
GRANT, P. T.....Widdicomb Bldg., OALR
ROBERTSON, F. D.....Ashton Bldg., OALR
ROLLER, L. A.....122 Monroe St., OALR
WELSH, D. E.....Powers Theater Bldg., OALR

IONIA

SEDGWICK, O. W..... OALR

IRON MOUNTAIN

BOYCE, G. H.....408 E. D. St., OALR

JACKSON

WINTER, G. E.....44 Sun Bldg., OALR

KALAMAZOO

FULKERSON, C. B....Kalamazoo National Bank Bldg., OALR
GRANT, F. E.....Kalamazoo National Bank Bldg., OALR
WILBUR, E. P.....Kalamazoo National Bank Bldg., OALR

MENOMINEE

ELWOOD, CALVIN R.....Spies Bldg., OALR

MT. CLEMENS

CROMAN, JOSEPH M. JR.....Washington Apts., OALR

ST. JOSEPH

MCDERMOTT, JOHN J.....St. Joseph Sanitarium, OALR

SAGINAW

MCKINNEY, ALEXANDER R.....Bearinger Bldg., OALR
ROGERS, A. S.....105 S. Jefferson Ave., OALR
WATSON, ROY S.....120 N. Michigan Ave., OALR

TRAVERSE CITY

HOLDSWORTH, FRANK.....206 Wilhelm Blk., OALR

MINNESOTA

DULUTH

BRIGGS, F. W.....Lyceum Bldg., ALR
COLLINS, HOMER.....New Jersey Bldg., OALR
TILDERQUIST, D. L.....7 E. Superior St., OALR
TURNBULL, F. M.....Providence Bldg., OALR
WINTER, JOHN A.....Fidelity Bldg., OALR

FERGUS FALLS

KITTLESON, THEODORE N.....101 Lincoln Ave., OALR

HIBBING

MORSMAN, L. W.....Congdon Bldg., OALR

MANKATO

JAMES, J. H.....National City Bank Bldg., OALR

MINNEAPOLIS

BEAUDOUX, H. A.....La Salle Bldg., Op.A
BISHOP, C. W.....La Salle Bldg., OALR
BROWN, E. J.....900 Nicollet Ave., OALR
CAMP, W. E.....Donaldson Bldg., OALR
CAMPBELL, ROBT. A.....La Salle Bldg., OALR
CLARK, H. S.....Syndicate Blk., OALR
DORGE, RICHARD I.....76 Seymour Ave., S. E., OALR
HANSEN, E. W.....Donaldson Bldg., OALR
LEE, J. W.....804 Besse Bldg., OALR
LEWIS, J. D.....La Salle Bldg., OALR
LITCHFIELD, J. T.....Yeats Bldg., OALR

LOOMIS, E. A.....Donaldson Bldg., OALR
MACINE, J. S.....Donaldson Bldg., OALR
MATTHEWS, JUSTUS .....Metropolitan Bldg., LR
MORSE, J. A.....Donaldson Bldg., OALR
MORTON, HOWARD McI.....Metropolitan Bk. Bldg., OALR
MURRAY, WM. R.....Nicollet Clinic, OALR
NEWHART, HORACE .....Donaldson Bldg., OALR
OBERG, C. M.....Syndicate Blk., OALR
PARKER, E. H.....La Salle Bldg., LR
PATTERSON, W. E.....La Salle Bldg., OALR
PHELPS, K. A.....La Salle Bldg., OALR
PRATT, F. J., JR.....Metropolitan Bldg., OALR
PRATT, J. A.....Metropolitan Bldg., OALR
REYNOLDS, J. S.....La Salle Bldg., OALR
SMITH, A. E.....Donaldson Bldg., OALR
STROUT, E. S.....Donaldson Bldg., OALR
STROUT, G. E.....Donaldson Bldg., OALR
WATSON, J. A.....Phys. and Surg. Bldg., OALR
WOOD, DOUGLAS.....Donaldson Bldg., OALR
WRIGHT, C. D.....Metropolitan Bldg., Op.A

ROCHESTER

BENEDICT, W. L.....Mayo Clinic,Op.
HEMPSTEAD, BERT F.....Mayo Clinic,ALR
LILLIE, H. I.....Mayo Clinic, ALR
NEW, G. B.....Mayo Clinic, LR
PRANGEN, A. DE H.....Mayo Clinic, Op.

ST. PAUL

BERRISFORD, PAUL D.....Lowry Bldg., OALR
BINGER, H. E.....Lowry Bldg., OALR
BOECKMAN, EGIL .....Lowry Bldg., OALR
BRAY, E. R.....1440 Lincoln Ave., OALR
BROWN, JOHN C.....Lowry Bldg., OALR
BURCH, F. E.....Hamm Bldg., A
CONNOR, CHAS. E.....Hamm Bldg., OALR
FOGARTY, CHAS. W.....1826 Summit Ave., OALR
FULTON, JOHN E.....728 Lowry Bldg., OALR
HOWARD, W. H.....137 N. Fairview Ave., OALR
LARSEN, CARL E.....Lowry Bldg., OALR
LEWIS, WILLIAM W.....836 Lowry Bldg., OALR
MALONEY, T. J.....Lowry Bldg., OALR
NELSON, L. A.....Lowry Arcade, Op.A
ROTHSCHILD, H. J.....Lowry Bldg., OALR
SHELLMAN, J. L.....Lowry Bldg., OALR
WHEELER, MERRITT W.....Lowry Bldg., OALR
WOLD, KARL C.....7th and Wacouta Sts., OALR

MISSISSIPPI

MERIDIAN

GUTHRIE, J. M.....Rosenbaum Bldg., OALR

OXFORD

GUYTON, B. S.....Carter Bldg., OALR

VICKSBURG

BELL, M. H.....First National Bank Bldg., OALR
EDWARDS, C. J.....1422 Wash., OALR
HOWARD, E. F.....First National Bank Bldg., ALR

MISSOURI

AURORA

O'DELL, TIMOTHY.....112 A Madison Ave., OALR

CARTHAGE

POST, WINIFRED B.....1380 S. Main St., OALR
POWERS, EVERETT.....Central National Bank Bldg., OALR

COLUMBIA

NOYES, G. L.....311 Hitt St., OALR
SNEED, C. M.....506 W. Broadway, OALR

KANSAS CITY

ALLEN, CHAS. E.....Waldheim Bldg., OALR
ALTRINGER, ARTHUR N.....Rialto Bldg., ALR
BEIL, J. W.....Argyle Bldg., OALR
BELLOWS, GEORGE E.....Rialto Bldg., Op.
BLAKESLEY, T. S.....Lathrop Bldg., OALR
CURDY, R. J.....Rialto Bldg., Op.
CURRAN, EDWIN J.....Waldheim Bldg., Op.
DAVIS, HARRY B.....Lathrop Bldg., OALR
FOSTER, HAL.....Chambers Bldg., ALR
HALL, EDW. P.....124 Hunter Ave., OALR
HOWARD, J. W.....Rialto Bldg., OALR
KEITH, W. E.....Waldheim Bldg., OALR
KIMBERLIN, J. W.....Rialto Bldg., Op.
LANING, J. H.....Lathrop Bldg., ALR
LEA, J. A.....Rialto Bldg., ALR
LEMOINE, A. N.....Lathrop Bldg., Op.
LEONARD, A. C.....Bryant Bldg., OALR
LICHTENBERG, J. S.....Rialto Bldg., Op.
LORIE, ALVIN.....Commerce Bldg., ALR
MAY, JAMES W.....800 Minnesota Ave., Op.
MILLER, HUGH.....Chambers Bldg., OALR
MOSS, H. E.....Rialto Bldg., OALR
MOTT, J. S.....Argyle Bldg., OALR
MYERS, J. L.....Lathrop Bldg., OALR
MCALESTER, A. W., JR.....Bryant Bldg., Op.
MCCORTY, V. W.....Rialto Bldg., ALR
PAINTER, A. M.....Sharp Bldg., ALR
PATTERSON, J. M.....Bryant Bldg., OALR
ROBERTS, S. E.....Waldheim Bldg., ALR

RUSSELL, E. L. ..... Argyle Bldg., OALR
SCHUTZ, W. H. ..... Bryant Bldg., OALR
SELLERS, L. M. ..... Argyle Bldg., ALR
SHOEMAKER, S. A. ..... U. S. Pub. Ser. Hosp., OALR
SHUMATE, D. L. ..... Shukert Bldg., OALR
THOMASON, H. E. ..... Rialto Bldg., OALR
TUREMAN, H. G. ..... Rialto Bldg., OALR
YAZEL, H. E. ..... Grand Ave. Temple, OALR

MOBERLY

DUTTON, C. K. ..... OALR

ST. JOSEPH

FORGRAVE, L. ROBERT ..... Logan Bldg., OALR
KENNEY, W. L. ..... Corby Forsee Bldg., OALR
MINTON, W. H. ..... Bartlett Bldg., OALR
PROUD, W. C. ..... Phys. and Surg. Bldg., OALR

ST. LOUIS

ALDEN, ARTHUR M. ..... 3858 Westminster Place, ALR
ARBUCKLE, M. F. ..... University Club Bldg., ALR
BALL, J. MOORES ..... 4500 Olive St., Op.
BARCK, CARL ..... Humboldt Bldg., Op.A
BARDENHEIER, F. G. A. ..... Frisco Bldg., Op.A
BLACK, W. D. ..... Metropolitan Bldg., OALR
BRYAN, W. M. C. ..... University Club Bldg., ALR
BURNS, S. S. ..... Humboldt Bldg., ALR
CHARLES, J. W. ..... Humboldt Bldg., Op.
DONNELL, NEWMAN R. ..... Metropolitan Bldg., Op.
DYER, CLYDE P. ..... Metropolitan Bldg., Op.
EWING, ARTHUR E. ..... Metropolitan Bldg., Op.
GOLDSTEIN, M. A. ..... 3858 Westminster Pl., ALR
GREEN, JOHN, JR. ..... Metropolitan Bldg., Op.
GUGGENHEIM, L. K. ..... Carleton Bldg., ALR
GUNDELACH, C. A. ..... 825 University Club Bldg., ALR
HARDESTY, J. F. ..... Humboldt Bldg., Op.
HARDY, W. F. ..... Metropolitan Bldg., Op.
HIGBEE, E. H. ..... Metropolitan Bldg., Op.
HOURN, G. E. ..... Univ. Club Bldg., ALR
JACOBS, M. W. ..... Carleton Bldg., Op.
JENNINGS, J. E. ..... Carleton Bldg., OALR
LAMB, HARVEY ..... Metropolitan Bldg., Op.
LEAVY, C. A. ..... Metropolitan Bldg., ALR
LOEB, H. W. ..... Humboldt Bldg., ALR
LUEDDE, W. H. ..... Metropolitan Bldg., Op.
LYMAN, HARRY W. ..... Carleton Bldg., ALR
PFINGSTEN, C. F. ..... 508 N. Grand Ave., ALR
POST, LAWRENCE ..... Metropolitan Bldg., Op.
POST, MARTIN H. ..... Metropolitan Bldg., Op.
PROETZ, ARTHUR W. ..... University Club Bldg., ALR
REIM, HUGO ..... Metropolitan Bldg., Op.
SAUER, W. E. ..... Humboldt Bldg., ALR

SCHOLZ, R. P.....Metropolitan Bldg., ALR
SCHWARTZ, F. A.....Metropolitan Bldg., Op.
SHANAN, W. E.....Metropolitan Bldg., Op.
SHOEMAKER, W. A.....Carleton Bldg., Op.
SLUDER, GREENFIELD.....3542 Washington Ave., ALR
TOOKER, C. W.....Carleton Bldg., Op.
WESTLAKE, S. V.....Humboldt Bldg., ALR
WIENER, MEYER .....Carleton Bldg., Op.
WOODRUFF, F. E.....Metropolitan Bldg., Op.

SEDALIA

LOVE, J. G.....Ilgenfritz Bldg., OALR
TITSWORTH, GUY.....111 W. 4th St., Op.

SPRINGFIELD

BAILEY, H.....840 Landers Bldg., OALR
COFFELT, THEODORE A.....Woodruff Bldg., OALR
KLINGER, THOS. O.....Landers Bldg., OALR

MONTANA

ANACONDA

DUNLAP, LAWRENCE G.....Electric Light Bldg., OALR

BUTTE

DAYTON, G. O.....Lewisohn Bldg., OALR
DONAVAN, J. A.....Phoenix Bldg., OALR
MORSE, A. W.....Phoenix Bldg., OALR
POTTER, PETER.....Granate and Alaska Sts., OALR

GREAT FALLS

COULTER, C. F.....First Natl. Bank Bldg., OALR

LEWISTOWN

DAVIS, FRANK C..... ALR

ROUNDUP

LEWIS, G. A.....29 Main St., OALR

NEBRASKA

BEATRICE

BAIRD, CHARLES G..... OALR
TUCKER, J. C..... OALR

COLUMBUS

CAMPBELL, C. H.....Columbus State Bk. Bldg., OALR

KEARNEY

FOX, CHAS. H.....2116 Central Ave., OALR

LINCOLN

BROOKS, E. B.....Terminal Bldg., OALR
COOK, S. E.....Richards Block, OALR

CURTIS, W. L.....612 Security Mutual Bank Bldg., OALR
DAYTON, W. L.....Funke Bldg., OALR
FURGASON, A. P.....Richards Block, OALR
HOMPES, J. J.....Security Mutual Bldg., OALR
SANDERSON, DAVID D.....Funke Bldg., OALR
WILLIAMS, J. P.....Funke Bldg., OALR
WOODWARD, J. M.....Richards Block
ZEMER, S. G.....Security Mutual Bldg., OALR

NORFOLK

GADBOIS, A. E.....421 Norfolk Ave., OALR
NELSON, LLOYD L..... OALR

OMAHA

BANISTER, J. M.....Brandeis Theater Bldg., OALR
CALLFAS, W. F.....Brandeis Bldg., ALR
FAIRCHILD, NORA M.....600 Brandeis Theater Bldg., Op.
GIFFORD, HAROLD .....Brandeis Bldg., Op.A
GIFFORD, SANFORD R.....702 Brandeis Theater Bldg., Op.
HANEY, W. P.....Brandeis Theater Bldg., OALR
ISAAC, DAVID.....Brandeis Theater Bldg., OALR
KNOVDE, A. R.....Omaha National Bank Bldg., ALR
KULLY, B. M.....631 City Natl. Bk. Bldg., ALR
LEMERE, H. B.....Brandeis Bldg., OALR
OWEN, F. S.....Brandeis Bldg., OALR
PATTON, J. M.....Brandeis Bldg., OALR
POTTER, GEORGE B.....402 City Natl. Bk. Bldg., OALR
POTTS, J. B.....Brandeis Bldg., AL
RUBENTHAL, CLARENCE .....Brandeis Bldg., OALR
TUCKER, J. C.....Brandeis Bldg., OALR
UREN, C. T.....Omaha National Bank Bldg., OALR
WHERRY, W. P.....Bankers Reserve Bldg., OALR

NEW HAMPSHIRE

NASHUA

NUTTER, C. F.....16 Amherst St., Op.

NEW JERSEY

ASBURY PARK

UPHAM, HELEN F.....305 Third Ave., OALR

ATLANTIC CITY

CHARLTON, C. C.....124 S. Illinois St., ALR
MCGIVERN, CHAS. S.....805 Pacific Ave., ALR
STICKNEY, O. D.....92 Pacific Ave., ALR

EAST ORANGE

BUVINGER, CHARLES W.....50 Washington St., OALR

JERSEY CITY

CHAMBERS, T. R.....15 Exchange Pl., OALR
PYLE, WALLACE.....18th and Exchange Pl., OALR

LONG BRANCH

CAMPBELL, W. K.....96 Third Ave., OALR

NEWARK

CHATTIN, J. F.....671 Broad St., Op.
HURFF, J. W.....86 Washington St., OALR
KEIM, WILLIAM F.....2 Lombardy St., OALR
O'CONNOR, F. O.....671 Broad St., OALR
ORTON, H. B.....671 Broad St., ALR
QUINBY, W. O'G.....14 James St., OALR
SHERMAN, E. S.....Wiss Bldg., OALR
ZEHNDER, C. A.....180 Fairmount Ave., OALR

ORANGE

EMERSON, LINN .....Metropolitan Bldg., OALR

RIVERSIDE

SCHURTER, M. A.....8 Morton Ave., OALR

TRENTON

ADAMS, CHARLES.....52 W. State St., OALR
CRANE, J. WELLINGTON.....128 Third St., ALR

WEST HOBOKEN

SACCO, A. G.....440 Clinton Ave., OALR

NEW MEXICO

ALBUQUERQUE

BREHEMER, HARRISON L.....521 Luna Blvd., OALR

NEW YORK

ALBANY

BEDELL, A. J.....344 State St., Op.A
BEGGS, W. F.....2 Lombardy St., OALR
DOWLING, J. I.....116 Washington Ave., OALR
HINMAN, EUGENE E.....146 Washington Ave., ALR
JUDGE, H. V.....228 State St., Op.

BINGHAMTON

WATSON, H. D.....151 Front St., OALR

BROOKLYN

ALBRIGHT, LT. P. M., M. C. U. Navy, U. S. Naval Hosp, ALR
ARROWSMITH, HERBERT.....170 Clinton St., ALR
HANCOCK, J. C.....135 Cambridge Pl., Op.
LOMBARDO, MELCHIORE.....186 Meserole St., Op.
MCCLELLAND, L. A.....78 McDonough St., ALR
MYERSON, MERVIN C.....184 Clinton St., ALR

BUFFALO

ANDREWS, H. D.....23 Allen St., Op.
BENNETT, A. G.....26 Allen St., Op.
BEYER, LOUIS J.....449 Delaware Ave., ALR
BLAAUW, E. E.....190 Ashland Ave., OALR
BOZER, HERRMANN, E.....438 Delaware Ave., ALR
BROWN, C. M.....510 Delaware Ave., ALR
COTT, C. C.....483 Delaware Ave., ALR
COTT, DELZON N.....1001 Main St., ALR
COWPER, H. W.....543 Franklin St., Op.
EDSON, RAY.....560 Delaware Ave., Op.
FAIRBAIRN, JOHN.....503 Delaware Ave., ALR
FLAGG, J. D.....473 Virginia Ave., Op.A
FRANCIS, L. M.....636 Delaware Ave., Op.
GLOSSER, HERBERT H.....448 Franklin St., OALR
HEALEY, J. F.....503 Delaware Ave., ALR
HOWE, LUCIEN.....520 Delaware Ave., Op.
HUBBARD, A. E.....372 Franklin St., Op.
LEWIS, F. P.....454 Franklin St., Op.
MARCH, CLARA A.....465 Ashland Ave., Op.
PHILLIPS, W. L.....759 Richmond Ave., Op.
RENNER, W. S.....341 Linwood Ave., ALR
SATTERLEE, R. H.....187 Delaware Ave., Op.
SERNOFFSKY, I.....37 Allen St., Op.
STARR, E. G.....523 Delaware Ave., Op.
WEED, HARRY M.....196 Lincoln Ave., Op.

CORTLAND

HIGGINS, REUBEN P.....20 Court St., OALR

DUNKIRK

JACKLE, A. F.....63 E. 4th St., Op.

GENEVA

SPENGLER, J. A.....423 Main St., OALR

ITHACA

BULL, E. L.....124 E. State St., OALR
KIRKENDELL, J. S.....315 N. Aurora, OALR
WILSON, R. C.....208 E. State St., OALR

JAMESTOWN

HOTCHKISS, W. W.....195 Forest Ave., OALR
REGER, H. S.....New Wellman Bldg., OALR

KENMORE

WURTZ, W. J. M.....2808 Delaware Ave., ALR

LIBERTY

DWORETSKY, JULIUS.....7 Law St., ALR

LOCKPORT

RINGUEBERG, EUGENE.....15 Main St., Op.

MT. VERNON

THOMSON, J. J.....3 Park Ave., OALR

NEW YORK CITY

AUERBACH, JULIUS.....120 W. 86th St., ALR
BALLIN, M. J.....108 E. 60th St., ALR
BELL, GEO. HUSTON.....40 E. 41st St., Op.
BERENS, CONRAD, JR.....9 E. 46th St., Op.
BOONE, JAMES L.....N. Y. Eye and Ear Infir., ALR
BRUDER, JOSEPH.....229 W. 79th St., OALR
CARTER, W. W.....24 E. 48th St., ALR
COHEN, MARTIN.....1 W. 85th St., OALR
COHN, FELIX.....31 E. 60th St., ALR
DAVIS, A. E.....47 E. 57th St., Op.
DAVIS, G. E.....42 W. 77th St., OALR
DIXON, G. S.....40 E. 41st St., R
DUANE, ALEXANDER.....139 E. 37th St., Op.
DUNNINGTON, JOHN H.....104 E. 40th St., Op.
FORBES, H. H.....40 E. 41st St., ALR
FREUDENTHAL, WOLF.....24 W. 88th St., ALR
FRIDENBERG, P. H.....38 W. 59th St., OALR
FRIEDMAN, DAVID.....44 W. 77th St., ALR
GATEWOOD, W. L.....15 E. 48th St., ALR
GLOGAU, OTTO.....64 E. 91st St., OALR
GOLDMAN, HARRY G.....57 W. 58th St., ALR
GOTTLIEB, M. J.....814 West End Ave., ALR
GRUSHLOW, ISRAEL.....271 Central Park, W., ALR
GUTTMAN, J.....987 Madison Ave., OALR
HASKINS, W. H.....40 E. 41st St., ALR
HAYS, H. N.....22 W. 74th St., ALR
HETRICK, LLEWELLYN E.....20 E. 53rd St., ALR
HOPKINS, W. E.....515 Park Ave., OALR
HURD, L. M.....39 E. 50th St., ALR
IMPERATORI, C. J.....17 E. 38th St., ALR
INGERMAN, SERGIUS.....418 Central Park, W., OALR
IRWIN, F. N.....114 E. 54th St., OALR
JACOBS, S. M.....1018 E. 163rd St., OALR
JARECKY, HERMAN.....138 W. 86th St., OALR
JOHNSON, THOMAS H.....30 W. 59th St., Op.
JUDD, H. B.....47 E. 57th St., ALR
KERRISON, P. D.....58 W. 56th St., ALR
KEY, B. W.....7 W. 49th St., Op.
KING, J. J.....40 E 41st St., ALR
KIRBY, DANIEL B.....30 W. 59th St., Op.
KNAPP, ARNOLD.....10 E. 54th St., Op.
KOPETZKY, S. J.....51 W. 73rd St., ALR
KRUG, E. F.....12 W. 44th St., OALR
LA VIGNE, A. A.....11 E. 48th St., OALR
LUBMAN, MAX.....135 E. 65th St., ALR
MAY, C. H.....698 Madison Ave., Op.
MAYBAUM, J. L.....12 E. 86th St., ALR

MAYER, EMIL.....40 E. 41st St., ALR
McCOY, JOHN.....157 W. 73rd St., ALR
MEIERHOF, E. L.....1140 Madison Ave., OALR
MITTENDORF, A. D.....399 Park Ave., Op.
MITTENDORF, W. K.....115 E. 53rd St., Op.A
NISSELSON, MAX.....616 Madison Ave., OALR
OPPENHEIMER, SEYMOUR.....45 E. 60th St., OALR
PHILLIPS, W. C.....40 W. 47th St., ALR
REESE, R. G.....50 W. 52nd St., Op.
SCHILLER, A. N.....225 West End Ave., ALR
SOMBERG, JOSEPH S.....45 E. 60th St., Op.
STURGES, L. F.....128 W. 87th St., LR
VOORHEES, IRVING W.....14 Central Park, W., ALR
WEEKS, JOHN E.....20 E. 53rd St., Op.
WEEKS, WEBB W.....20 E. 53rd St., Op.
WHEELER, J. M.....30 W. 59th St., Op.
WIENER, ALFRED.....550 Park Ave., OALR
YANKAUER, SIDNEY.....616 Madison Ave., ALR

NIAGARA FALLS

PRICE, N. W.....445 Third St., OALR
ROOKER, A. L.....1347 Michigan Ave., ALR

NYACK-ON-HUDSON

TOMS, S. W. S..... ALR

OLEAN

TINDOLPH, LEA W.....First Natl. Bk. Bldg., OALR

ONEIDA

CROCKETT, R. L..... Op.

POUGHKEEPSIE

KRIEGER, W. A.....36 Market St., OALR

ROCHESTER

BARBER, FRANK.....75 S. Fitzhugh St., Op.
CARROLL, G. G.....614 Main St., W., OALR
CLARK, L. H.....337 Monroe Ave., OALR
LERNER, MACY L.....332 Park Ave., Op.
McDOWELL, N. D.....275 Alexander St., OALR
MORRIS, A. G.....41 Gibbs St., OALR
SHAPERO, I. M.....365 East Ave., OALR

SYRACUSE

BROWN, G. M.....802 University Bldg., ALR
BRITTEN, G. S.....University Blk., OALR
BRUST, H. O.....720 S. Crouse Ave., ALR
FOWLER, S. R.....University Bldg., ALR
KAUFFMAN, ARNOLD B.....Gurney Bldg., ALR
KLINE, H. G.....University Blk., ALR

TROY

SULZMAN, F. M.....1831 Fifth Ave., OALR

UTICA

BEATTIE, W. HENRY.....252 Genesee St., OALR

NORTH CAROLINA

ASHEVILLE

BRIGGS, H. H.....73 Haywood St., OALR
GREENE, JOSEPH B.....Haywood Bldg., ALR

CHARLOTTE

MATHESON, J. P.....511 Independence Bldg., OALR
PEELER, C. N.....W. 7th St., OALR
SLOAN, HENRY L.....Independence Bldg., Op.
WHISNANT, A. M.....Realty Bldg., OALR

GREENSBORO

REAVES, W. P.....117 W. Sycamore St., OALR

NEW BERN

DANIELS, R. L.....Elks' Temple, OALR

RALEIGH

WRIGHT, J. B.....Citizens Natl. Bank Bldg., OALR

SALISBURY

BRAWLEY, R. V.....Wallace Bldg., OALR

WILMINGTON

MURPHY, J. G.....Murchison Bldg., OALR

WINSTON-SALEM

DAVIS, THOMAS W.....O'Hanlon Bldg., OALR

NORTH DAKOTA

FARGO

RINDLAUB, ELIZABETH P.....De Lendrecie Blk., OALR
RINDLAUB, J. H.....De Lendrecie Blk., OALR
RINDLAUB, M. P., JR.....De Lendrecie Blk., OALR

MINOT

MCCANNELL, A. D.....Colcord Bldg., OALR

WAHPETON

LANCASTER, WILSON.....310 Dakota Ave., OALR
RICE, C. P.....Sterns Bldg., OALR

OHIO

AKRON

BROWN, L. E.....2nd Natl. Bank Bldg., OALR
MOORE, T. K.....Hamilton Bldg., OALR
STEVENSON, D. W.....165 E. Market St., OALR

ALLIANCE

KING, G. L.....437 E. Market St., OAR

BELLEFONTAINE

BUTLER, ROBERT H.....227 W. Columbus Ave., OALR
HARBERT, J. P.....Canby Blk., OALR

CANTON

SCHILD, E. H.....9th and Cleveland, Op.A

CINCINNATI

ALLEN, S. E.....11½ E. 8th St., OALR
GOODYEAR, HENRY M.....8 E. 8th St., ALR
HANS, C. L.....19 W. 7th St., OALR
HARRIS, W. C.....Groton Bldg., ALR
HINNEN, G. A.....1343 Delta Ave., OALR
IGLAUER, SAMUEL .....Livingston Bldg., ALR
JONES, CHARLES C.....19 Garfield Pl., ALR
KING, C.....Union Central Bldg., Op.
KING, EDWARD.....605 Traction Bldg., ALR
LAMB, F. W.....7th and Vine Sts., OALR
MITHOEFER, WILLIAM.....19 Garfield Pl., ALR
MURPHY, W. E.....7th and Race Sts., OALR
PHINNEY, F. D.....22 W. 7th St., OALR
RAY, VICTOR.....19 W. 7th St., OALR
SIEGEL, F. X.....2700 Union Central Bldg., OALR
STANBERRY, HENRY .....Doctors Bldg., Op.AR
STEVENSON, ROBERT.....11½ E. 8th St., ALR
STEWART, T. M.....Union Trust Bldg., OALR
STRICKER, LOUIS .....Groton Bldg., Op.
TANGEMAN, H. F.....Central Bldg., ALR
THOMPSON, E. H.....Vindonissa Bldg., 19 W. 7th St., OALR
URNER, M. H.....Union Central Life Bldg., OALR
VAIL, D. T.....24 E. 8th St., Op.
VAIL, H. H.....24 E. 8th St., ALR

CLEVELAND

ABBOTT, W. J.....8314 Euclid Ave., OALR
BRUNER, ABRAM B.....Guardian Bldg., Op.
BRUNER, W. E.....Guardian Bldg., Op.
BURKE, T. A.....Rose Bldg., OALR
COGAN, J. E.....Rose Bldg., OALR
CUTLER, FRANKLIN E.....1025 Schofield Bldg., ALR
FORSYTHE, S. T.....1011 Euclid Ave., OALR
INGERSOLL, J. M.....Osborn Bldg., ALR
KENDALL, M. R.....Osborn Bldg., ALR
LARGE, S. H.....2323 Prospect Ave., ALR
LENKER, J. N.....Osborn Bldg., ALR
METZ, R. B.....Guardian Bldg., Op.
METZENBAUM, MYRON .....Rose Bldg., ALR
MONSON, S. H.....Ainsfield Bldg., OALR

MUSSUN, WILLIAM G.....Osborn Bldg., ALR
McDONALD, C. L.....Ainsfield Bldg., OALR
NELSON, C. F.....Schofield Bldg., OALR
PITKIN, C. E.....Osborn Bldg., ALR
PRENDERGAST, D.....1110 Euclid Ave., OALR
QUITTNER, S. S.....3912 Prospect Ave., OALR
RIEGER, WALTER H.....Hanna Bldg., ALR
RUST, E. G.....Hanna Bldg., Op.A
SHACKLETON, W. E.....2323 Prospect Ave., Op.
SHIRAS, H. H.....Guardian Bldg., Op.
SIMONDS, O. F.....Hanna Bldg., Op.
STOTTER, A. L.....1148 Euclid Ave., OALR
STOTTER, JAMES.....1148 Euclid Ave., OALR
TRIPP, I. C.....Rose Bldg., OALR
TUCKERMAN, W. C.....Osborn Bldg., Op.
TUCKERMAN, W. H.....Osborn Bldg., OALR
WAUGH, J. M.,
Cleveland Clinic, Euclid Ave. at 93rd St., ALR
WOLFENSTEIN, LEO .....Rose Bldg., Op.

COLUMBUS

BEATTY, H. G.....150 E. Broad St., OALR
BROWN, J. E.....370 E. Town St., OALR
CLARK, I. G.....188 E. State St., OALR
HAUER, A. M.....327 E. State St., OALR
PROUT, A. W.....370 E. Town St., OALR
SILBERNAGEL, C. E.....283 E. State St., OALR
SULZER, G. A.....200 E. State St., OALR
THOMAS, F. W.....74 S. 5th St., OALR
TIMBERMAN, ANDREW.....525 City Bk. Bldg., OALR
WOLFE, A. C.....350 E. State St., ALR
WRIGHT, J. W.....Central National Bank Bldg., Op.

DAYTON

BINKLEY, R. S.....Fidelity Med. Bldg., OALR
BLACKBURN, W. J.....Reibold Bldg., OALR
DUTROW, H. V.....1040 Fidelity Medical Bldg., OALR
FARMER, A. G.....Fidelity Medical Bldg., OALR
FOUTS, JOHN D.....Fidelity Medical Bldg., OALR
HARRIS, H. B.....Fidelity Medical Bldg., OALR
KILBOURNE, PERLEY H.....Fidelity Medical Bldg., OALR
MILLETTE, J. W.....Reibold Bldg., OALR

ELYRIA

GILL, GEORGE.....146 Middle Ave., OALR

IRONTON

DUNN, O. B.....Fourth and Railroad Sts.

LIMA

STUEBER, F. G.....Main and North Sts., OALR
STUEBER, PAUL J.....209 W. North St., OALR

LORAIN

MONOSMITH, O. B.....424 Broadway, OALR

MANSFIELD

GARBER, J. M.....48 Park Ave., OALR
WISE, R. C.....News Bldg., OALR

MARIETTA

SAUER, W. W.....Box 416, OALR

MARION

BRICKLEY, D. W.....196 W. Center St., OALR

MIDDLETOWN

WILLIAMS, WALTER H.....OALR

NEWARK

HATCH, C. B.....3 W. Church St., OALR

SALEM

HILL, A. J.....301 Citizens Natl. Bank Bldg., OALR

SANDUSKY

BLISS, C. B.....411 Columbus Ave., OALR

SPRINGFIELD

EASTON, J. C.....Fairbanks Bldg., OALR
HARTLEY, F. A.....Fairbanks Bldg., OALR
HOGUE, D. W.....Fairbanks Bldg., OALR
MINOR, C. L.....Fairbanks Bldg., OALR

STEUBENVILLE

GOURLEY, G. F.....Sinclair Bldg., OALR

TIFFIN

PORTER, E. H.....85 Madison St., OALR

TOLEDO

ALDER, F. W.....Toledo Medical Bldg., OALR
ALDERDYCE, W. W.....Nasby Bldg., OALR
DENMAN, IRA O.....Ohio Bldg., OALR
HOBART, BERTHA K.....Produce Exchange Bldg., OALR
JACOBI, FRANK.....Colton Bldg., OALR
KELLER, T. F.....The Spitzer, OALR
KING, CHARLES R.....Spitzer Bldg., OALR
LUKENS, CHARLES.....218 Michigan Ave., OALR
O'BRIEN, C. S.....Colton Bldg., Op.
SNYDER, W. H.....211 Ontario St., OALR
STEINFELD, A. L.....234 Michigan St., OALR

XENIA

MADDEN, REED.....Allen Bldg., OALR
SHIELDS, L.....Allen Bldg., ALR

YOUNGSTOWN

BEARD, HAROLD J.....Dalton Bldg., OALR
GIBSON, R. D.....Dollar Savings and Trust Bldg., OALR
HARTZELL, S. M.....Dollar Bank Bldg., OALR

ZANESVILLE

BARON, F. S.....Peoples Bank Bldg., OALR
CULBERTSON, L. R.....Masonic Temple, OALR

OKLAHOMA

BARTLESVILLE

KISER, J. D.....Central Natl. Bank Bldg., OALR

GUTHRIE

BARKER, CHAS. B.....224½ W. Oklahoma Ave., OALR

MCALISTER

DAVIS, J. E.....Kress Bldg., OALR

MUSKOGEE

FULLENWIDER, C. M.....Barnes Bldg., OALR

OKLAHOMA CITY

DIXON, W. E.....706 1st Natl. Bank Bldg., OALR
FERGUSON, E. I.....607 1st Natl. Bank Bldg., OALR
GUTHRIE, A. L.....Amer. Natl. Bank Bldg., OALR
MCHENRY, D. D.....Colcord Bldg., OALR
NEWTON, L. A.....Colcord Bldg., OALR
TODD, H. C.....Colcord Bldg., OALR

TULSA

BRASWELL, C. J.....726 Mayo Bldg., OALR
COOK, W. ALBERT.....506 Palace Bldg., OALR
ROTH, A. W.....Palace Bldg., OALR

OREGON

LA GRANGE

BOUVY, HARRY.....New Foley Bldg., OALR

PORTLAND

AINSLIE, GEORGE.....Oregonian Bldg., OALR
BRUERE, G. E.....Journal Bldg., OALR
DAVIS, RALPH F.....Selling Bldg., OALR
FENTON, RALPH A.....616 Journal Bldg., OALR
HENTON, GEORGE EARL.....Morgan Bldg., OALR
JOHNSTON, WILSON.....Stevens Bldg., OALR
KIEHLE, F. A.....Corbett Bldg., OALR
KISTNER, F. B.....Stevens Bldg., ALR
MCCOOL, JOSEPH L.....Stevens Bldg., Op.

PENNSYLVANIA

ALTOONA

GLOVER, S. P.....1118 12th Ave., OALR

BEAVER

SCROGGS, J. J.....218 E. 3rd St., OALR

BETHLEHEM

CHASE, WALTER D.....230 E. Broad St., ALR

BRADFORD

ASH, GARRETT G.....1 Main St., OALR

BUTLER

DOANE, L. L.....Reiber Bldg., OALR
HAZLETT, L. R.....117 S. Main St., OALR

CHARLEROI

FERMAN, JOHN W.....Stahlman Bldg., OALR
STAHLMAN, F. C.....Stahlman Bldg., OALR

CHESTER

CROSS, G. H.....525 Welsh St., Op.

CYNWYD

TUCKER, GABRIEL E.....335 Trevor Lane, ALR

ERIE

DENNIS, D. N.....221 W. 9th St., Op.A
DENNIS, E. P.....311 W. 10th St., OALR
DUNN, I. J.....Masonic Temple, OALR
FUST, J. H.....138 W. 9th St., OALR
HEARD, C. F.....Masonic Temple, OALR
JACKSON, J. D.....2324 W. 8th St.
RUSSELL, J. A.....206 Masonic Temple, ALR
SCHLINDWEIN, G. W.....138 W. 9th St., OALR
SHREVE, O. M.....162 W. 8th St., OALR
WRIGHT, KATHERINE L.....247 W. 8th St., ALR

FRANKLIN

JOBSON, G. B.....Printz Bldg., OALR

GREENSBURG

FAIRING, JOHN W.....204 Tribune Bldg., OALR
MCKEE, C. W.....Coulter Bldg., OALR

HARRISBURG

DAILY, G. L.....713 N. 3rd St., OALR
FARNSLER, H. H.....1438 Market St., OALR
GRACEY, GEORGE F.....209 State St., OALR
PARK, J. W.....32 N. 2d St., OALR
REBUCK, C. S.....412 N. 3d St., OALR

HAZLETON

REICHE, O. C.....328 W. Broad St., OALR

HOMESTEAD

BROWN, W. E.....8th Ave. and Amity St., OALR

HUNTINGDON

SEARS, W. H.....514 Penn St., OALR

JOHNSTOWN

BARKER, O. G. A.....Johnstown Trust Bldg., Op.
HARRIS, CLARENCE M.....Johnstown Trust Bldg., OALR
RUSH, CALVIN C.....342 Main St., OALR

MIDDLETOWN

GEORGE, H. W.....19 N. Union St., Op.

NEW BRIGHTON

NORTON, ROY R.....Masonic Bldg., OALR

NEW KENSINGTON

MCCONNELL, T. E.....Alter Bldg., OALR
STEIM, JOSEPH M.....1st Natl. Bk. Bldg., Op.

PHILADELPHIA

ALEXANDER, GEORGE J.....1831 Chestnut St., ALR
ANNON, WALTER T.....259 S. 17th St., ALR
APPLEMAN, L. F.....308 S. 16th St., Op.
BABBITT, JAMES A.....2117 Chestnut St., ALR
BALDWIN, KATE W.....1117 Spruce St., ALR
BALENTINE, P. L.....1524 Chestnut St., Op.
BOEHRINGER, H. W.....1811 S. 22nd St., Op.
BROWN, SAMUEL HORTON.....1901 Mt. Vernon St., Op.
BRUMM, SETH A.....Stock Exchange Bldg., ALR
BUTLER, MARGARET F.....1831 Chestnut St., ALR
BUTT, M. M.....2045 Chestnut St., Op.
CLERF, LOUIS H.....128 S. 10th St., ALR
COATES, G. M.....1736 Pine St., ALR
COHEN, SAMUEL.....1626 Spruce St., ALR
CRAMPTON, G. S.....1700 Walnut St., Op.
CREIGHTON, W. J.....1905 Chestnut St., Op.
CROSKEY, J. W.....1909 Chestnut St., OALR
DAVIS, J. L.....135 S. 18th St., ALR
DEICHLER, LYN WALTER.....2028 Chestnut St., OALR
DINTENFASS, HENRY.....1714 Pine St., ALR
EARL, HARRY D.....3717 Spruce St., ALR
ERSNER, M. S.....1729 Pine St., ALR
FELT, C. L.....N. E. Cor. Chestnut & 20th Sts., ALR
FOX, L. W.....303 S. 17th St., Op.
GLEASON, E. B.....2033 Chestnut St., ALR
GODDARD, H. M.....1531 Spruce St., ALR
GRISCOM, J. M.....1925 Chestnut St., Op.
HAWMAN, E. G.....4051 Broad St., OALR
HEED, C. R.....1205 Spruce St., Op.
HERBERT, A. W.....119 E. Lehigh Ave., OALR
HOLLOWAY, T. B.....1819 Chestnut St., Op.
HUSIK, DAVID N.....1610 Spruce St., ALR

JACKSON, C.....128 S. 10th St., L
KAUFMAN, A. S.....1923 Spruce St., ALR
KEELER, J. C.....254 S. 16th St., ALR
KRAUSS, FREDERICK.....1701 Chestnut St., OALR
LAESSLE, H. A.....The Lenox, 13th and Spruce Sts., ALR
LAWRENCE, GRANVILLE A.....Medical Arts Bldg., Op.
LEWIS, FIELDING O.....259 S. 17th St., ALR
LOFTUS, J. E.....605 Medical Arts Bldg., ALR
LOVE, LOUIS F.....1207 Spruce St., Op.
LUKENS, R. McD.....1308 Hunting Park Ave., ALR
MACFARLIN, DOUGLAS.....1805 Chestnut St., OALR
MACKENZIE, ALICE V.....1724 Spruce St., OALR
MACKENZIE, GEORGE W.....1724 Spruce St., ALR
MILLER, E. B.....2008 Walnut St., Op.
MITCHELL, E. K.....704 Lehigh Ave., ALR
MOORE, W. F.....255 S. 16th St., ALR
OLSHO, SYDNEY L.....235 S. 15th St., OALR
O'REILLY, C. A.....1901 Chestnut St., ALR
PETER, LUTHER C.....N. E. Cor. Chestnut and 20th Sts., Op.
PONTIUS, P. J.....1831 Chestnut St., OALR
POSEY, WM. CAMPBELL.....21st and Chestnut Sts., Op.
RADCLIFFE, McCLUNEY.....1906 Chestnut St., Op.
REESE, WARREN S.....230 S. 21st St., Op.
RIDPATH, R. F.....1928 Chestnut St., ALR
ROMMELL, J. C.....4501 N. Broad St., OALR
SARTAIN, PAUL J.....2006 Walnut St., Op.
SCARLETT, HUNTER W.....2228 Locust St., Op.
SCHATZ, H. A.....2035 Chestnut St., ALR
SCHNEIDEMAN, T. B.....Professional Bldg., Op.
SCHWEINITZ, G. E. DE.....1705 Walnut St., Op.
SCHWENK, P. N. K.....1417 N. Broad St., Op.
SENER, W. J.....130 S. 18th St., OALR
SHEMELEY, W. G., JR.....1724 Spruce St., OALR
SKILLERN, R. H.....1928 Chestnut St., LR
SMITH, S. McC.....1420 Spruce St., ALR
SPENCER, W.....1830 S. Rittenhouse Sq., OALR
STILES, C. M.....1831 Chestnut St., Op.
STOUT, PHILIP S.....Medical Arts Bldg., ALR
STROUSE, FRED M.....1301 Spruce St., ALR
SWEET, W. M.....1205 Spruce St., Op.
WAGERS, A. J.....The Lenox, 13th and Spruce Sts., ALR
WELCH, R. S. G.....League Island Nav. Hosp., OALR
WILLIAMS, H. J.....5908 Green St., Germantown, ALR
ZIEGLER, S. L.....1625 Walnut St., Op.
ZENTMAYER, WILLIAM.....1506 Spruce St., Op.

PITTSBURGH

BEANE, GEORGE W.....Jenkins Arcade Bldg., OALR
BERNATZ, C. F.....Park Bldg., Op.
BLAIR, W. W.....Diamond Bank Bldg., Op.

CARSON, W. E.....Jenkins Arcade Bldg., Op.
CRAIGHEAD, NANCY B.....Jenkins Arcade Bldg.
CURRY, G. E.....Westinghouse Bldg., Op.
DAY, EWING W.....Westinghouse Bldg., ALR
DICKINSON, B. M...5634 Stanton Ave., E. Liberty Sta., ALR
FRANK, A. C.....Diamond Bank Bldg., ALR
GROSS, JULIUS E.....Jenkins Arcade Bldg., OALR
GUERINOT, A. J.....Jenkins Arcade Bldg., OALR
HECKEL, E. B.....Jenkins Arcade Bldg., Op.
KNEEDLER, G. C.....Jenkins Arcade Bldg.
KREBS, A.....Jenkins Arcade Bldg., Op.
LINN, J. G.....Highland Bldg., OALR
MARKEL, J. C.....Westinghouse Bldg., Op.
MARSHALL, WATSON.....Diamond Bank Bldg., ALR
McCREADY, J. H.....Empire Bldg., ALR
McKEE, GEORGE J.....1118 Westinghouse Bldg., ALR
MacLACHLAN, A. A.....May Bldg., ALR
MEANOR, W. C.....Westinghouse Bldg., Op.
MURDOCH, J. F.....Bessemer Bldg., Op.
PATTERSON, E. J.....Westinghouse Bldg., ALR
SEEGMAN, SIMON.....Jenkins Arcade Bldg., ALR
SHUMAN, G. H.....Park Bldg., Op.
SIMPSON, JOHN R.....1005 Westinghouse Bldg., ALR
SMITH, S. S.....Jenkins Arcade Bldg., Op.
STIEREN, EDWARD.....Union Trust Bldg., Op.
STIMSON, GEO. W.....Jenkins Arcade Bldg., ALR
STURM, S. A.....Jenkins Arcade Bldg., Op.
TAYLOR, MARTIN C.....415 Warrington Ave., Op.
TODD, G. C.....307 Westinghouse Bldg., ALR
TURNER, HUNTER H.....Jenkins Arcade Bldg., Op.
VAN KIRK, V. E.....Union Arcade Bldg., Op.
WEIMER, EDGAR S.....332 Lehigh Ave., Op.
WEISSER, E. A.....Empire Bldg., Op.
WIBLE, E. E.....Diamond Bank Bldg., Op.

READING

STOCKLER, J. A.....332 N. 5th St., OALR

RIDGEWAY

McALISTER, J. C.....10 S. Mill Ave., OALR

SCHUYLKILL HAVEN

MOORE, GEORGE H.....200 Main St., OALR

SCRANTON

CORSER, J. B.....Scranton Private Hospital, OALR
CURTIN, EUGENE A.....Connell Bldg., OALR
FREY, C. L.....529 Pine St., OALR
SULLIVAN, JOHN J., JR.....Traders Bk. Bldg., ALR

SUNBURY

BECKER, H. M.....49 S. 4th St., OALR

UPPER DARBY

SHANNON, C. E.....104 Copley Rd., Op.

WARREN

BALL, M. V.....214 Penn Ave., W., OALR

WASHINGTON

McKENNAN, J. W.....Washington Trust Bldg., Op.
McMURRAY, J. B.....Washington Trust Bldg., OALR

WILKES-BARRE

BUCKMAN, E. V.....70 S. Franklin St., OALR
CARR, G. W.....54 S. Franklin St., OALR
KISTLER, O. F.....43 N. Franklin St., Op.A
McLAUGHLIN, P. A.....73 S. Washington St., OALR
SCHAFFERT, N. L.....57 S. Washington St., OALR
TAYLOR, LEWIS H.....83 S. Franklin St., OALR

YORK

KLINEDIENST, J. F.....220 S. George St., OALR

RHODE ISLAND

NEWPORT

JACOBY, D. P. A.....106 Touro, Op.AL

PROVIDENCE

ADAMS, F. M.....224 Thayer St., ALR
CAPRON, F. P.....118 Angell St., OALR
HARVEY, N. D.....114 Waterman Ave., OALR
LEECH, J. W.....111 Broad St., OALR
MESSINGER, H. C.....170 Broad St., Op.
MUNCY, W. M.....25 Waterman St., OALR
PORTER, L. B.....117 Waterman St., OALR

SOUTH CAROLINA

CHARLESTON

KOLLOCK, C. W.....86 Wentworth, OALR
TOWNSEND, JOHN F..85 Wentworth,Cor. St. Phillips St., OALR

COLUMBIA

WHALEY, E. M.....1430 Blanding St., OALR

GREENVILLE

CARPENTER, E. W.....201 E. North St., ALR
MAULDIN, L. O.....Wallace Bldg., OALR

ROCK HILL

TWITTY, W. C.....Peoples Natl. Bk. Bldg., OALR

SOUTH DAKOTA

ABERDEEN

ALWAY, ROBERT D.....423 S. Lincoln St., OALR

HURON

GROSVENOR, L. N.....378 Dakota St., OALR

MITCHELL

BOBB, E. V.....First Natl. Bk. Bldg., OALR

RAPID CITY

WALSH, JOSEPH M..... OALR

SIOUX FALLS

GREGG, J. B.....Security Natl. Bank Bldg., OALR
PARSONS, J. G.....Paulton Bldg., OALR
PUTNAM, F. I.....708 Sioux Falls Natl. Bk. Bldg., OALR

WATERTOWN

JOHNSON, A. E.....Citizens Bk. Bldg., OALR

YANKTON

HOHF, JULIUS.....Clinic Bldg., OALR

TENNESSEE

CHATTANOOGA

HACKNEY, F. J.....630 Volunteer Bldg., OALR
HOGSHEAD, J. M.....602 Georgia Ave., OALR
JOHNSON, O. H.....630 Volunteer Bldg., OALR

DYERSBURG

BREWER, JOHN D.....618 Main St., OALR

JACKSON

DANCY, A. B.....First National Bank Bldg., OALR

KNOXVILLE

KINCAID, J. H.....Ross Flats, OALR
REAVES, CHAS. R.....Box No. 187, OALR

MEMPHIS

BLASSINGAME, CHARLES D.....Exchange Bldg., ALR
BLUE, J. B.....Exchange Bldg., OALR
ELLETT, E. C.....Exchange Bldg., Op.
FARRINGTON, P. M.....1473 Goodbar Ave., ALR
HARRIS, W. ROBIN.....Exchange Bldg., ALR
LEVY, LOUIS.....Bank of Commerce Bldg., OALR
LEWIS, A. C.....Exchange Bldg., OALR
MINOR, H. F.....Randolph Bldg., OALR
McKINNEY, R.....Bk. of Commerce and Trust Bldg., ALR
SAVAGE, G. H.....Central Bank Bldg., OALR
SHEA, J. J.....Exchange Bldg., OALR
SIMPSON, W. L.....Exchange Bldg., OALR
STANFORD, J. B.....Exchange Bldg., OALR

NASHVILLE

CAYCE, E. B.....Hitchcock Bldg., OALR

CULLOM, MARVIN McT.....208 Hitchcock Bldg., OALR
KENNON, WILLIAM G.....Lambuth Bldg., OALR
WOOD, HILLIARD.....Independence Bldg., OALR

SOUTHSIDE

VAUGHAN, G. E.....Franklin St., OALR

TEXAS

AUSTIN

BOERNER, MORRIS H Scarborough Bldg., OALR

BROWNWOOD

ANDERSON, W. B.....507 Main Ave., OALR

CUERO

DUCKWORTH, GUILFORD M.....Buchel Bldg., OALR

DALLAS

BETTISON, D. L.....502 Wilson Bldg., OALR
CARPENTER, E. R.....Medical Arts Bldg., OALR
CARY, E. H.....Southwestern Life Bldg., OALR
DECHERD, H. B.....Wilson Bldg., OALR
McREYNOLDS, J. O.....Trust Bldg., OALR
SEAY, D. E.....Dallas Co. State Bk. Bldg., OALR

EL PASO

IRVIN, E. H.....321 W. Rio Grande St., OALR
SCHUSTER, STEPHEN A.....1st Natl. Bk. Bldg., ALR

FORT WORTH

HOWARD, E. L.....First Natl. Bank Bldg., OALR
JOYES, CRITTENDEN..Farm. & Merch. Natl. Bank Bldg., OALR
SCHENK, C. P.....Tex. State Bk. Bldg., OALR

HOUSTON

FOSTER, J. H.....1300 Walker Ave., ALR
HADEN, H. C.....913 Union Natl. Bk. Bldg., OALR
ISRAEL, N. E.....Carter Bldg., OALR
ISRAEL, SIDNEY .....Carter Bldg., OALR
RALSTON, W. W.....1300 Walker Ave., Op.

LONGVIEW

HURST, V. REEVES..... OALR

SAN ANTONIO

ADAMS, ELDRIDGE S.....Moore Bldg., OALR
ATKINSON, D. T.....1515 Russell Bldg., OALR
BURLESON, J. H.....Central Trust Bldg., OALR
GWINN, G. E.....Hicks Bldg., OALR
MOSS, R. E.....Hicks Bldg., OALR

TEMPLE

McREYNOLDS, G. S.....State Bank Bldg., OALR
WOODSON, J. M.....City National Bank Bldg., OALR

TEXARKANA

FULLER, T. E.....Texarkana Natl. Bank Bldg., OALR
MANN, R. H. T.....State National Bank Bldg., OALR

TYLER

VAUGHAN, E. H.....113½ W. Ferguson St., OALR

WACO

AYNSWORTH, HORACE.....Amicable Bldg., OALR

UTAH

OGDEN

HETZEL, C. C.....Eccles Bldg., OALR

PROVO

MERRILL, H. G.....35 East, 12 South St., OALR

SALT LAKE CITY

DONOHER, W. D.....Boston Bldg., OALR
HAMPTON, R. H.....Boston Bldg., OALR
LINDSAY, D. M.....Boston Bldg., OALR
SNOW, LESLIE W.....Deseret Bank Bldg., OALR
STAUFFER, FREDERICK.....Deseret Bank Bldg., OALR

VERMONT

BURLINGTON

ARNOLD, F. J.....182 Pearl St., OALR
BROWN, E. T.....30 Church St., OALR

RUTLAND

MARSHALL, GEORGE G.....Gryphon Bldg., OALR

VIRGINIA

RICHMOND

HILL, EMORY.....Professional Bldg., Op.
HUGHES, THOMAS E.....Medical Arts Bldg., ALR
MILLER, C. M.....Stuart Circle Hospital, OALR

ROANOKE

GARRETT, J. R.....Strickland Bldg., OALR
GILL, E. G.....McBain Bldg., ALR

WINCHESTER

McGUIRE, H. H.....105 N. Braddock St., OALR

WASHINGTON

ABERDEEN

MAXEY, E. E.....4 Electric Bldg., OALR

BELLINGHAM

VAN KIRK, FRANK J.....Bellingham Natl. Bk. Bldg., OALR

SEATTLE

ADAMS, F. W.....Roosevelt Clinic, OALR
CHASE, E. FRANK.....Cobb Bldg., OALR
DOWLING, J. F.....Cobb Bldg., OALR
HOFFMAN, W. F.....Leary Bldg., OALR
SEELYE, W. K.....Amer. Bk. Bldg., OALR
STILLSON, HAMILTON.....Seabord Bldg., OALR
WÜRDEMANN, H. V.....Cobb Bldg., OALR

SPOKANE

ROTT, O. M.....Paulsen Bldg., OALR
VEASEY, C. A.....Paulsen Bldg., OALR

TACOMA

CAMMERON, WM. G.....Natl. Realty Bldg., OALR
HOWE, A. W.....Puget Sound Bk. Bldg., OALR
WHEELER, E. C.....Tacoma Clinic, OALR

WEST VIRGINIA

CHARLESTON

COVINGTON, L. C.....217½ Capitol Ave., OALR
YOUNG, H. H.....Morrison Bldg., OALR

CLARKSBURG

LOUCHERY, D. C.....214 W. Main St., OALR

FAIRMONT

JOHNSON, H. R.....512 Walnut Ave., OALR

HUNTINGTON

MOORE, T. W.....1050 Fifth Ave., OALR

MORGANTOWN

WYLIE, CHARLES B.....293 High St., OALR

WHEELING

FAWCETT, IVAN.....Schmulbach Bldg., OALR
OESTERLING, H. E.....Wheeling Steel Corp. Bldg., OALR
TOMASSENE, RAYMOND A..Wheeling Steel Corp. Bldg., OALR

WISCONSIN

APPLETON

BROOKS, E. H.....808 College Ave., OALR
RECTOR, A. E.....812 College Ave., OALR

EAU CLAIRE

PAYNE, A. L.....Opera House Bldg., OALR

GREEN BAY

BELLIN, JOSEPH.....322 N. Washington St., OALR
COMEE, W. C.....Bellin Bldg., OALR
NADEAU, E. G.....Bellin-Buchanan Bldg., OALR

JANESVILLE

LINTLEMAN, F. R.....Hayes Blk., OALR

KENOSHA

ASHLEY, T. W.....466 Park Ave., OALR

LA CROSSE

BRADFIELD, J. A. L.....State Bank Bldg., OALR
DOUGLAS, F. A.....State Bank Bldg., OALR
EDWARDS, W. A.....329 Main St., OALR
SMITH, D. S.....State Bank Bldg., OALR

MADISON

DWIGHT, C. G.....Pioneer Bldg., OALR

MANITOWOC

ANDREWS, M. P.....Dempsey Bldg., OALR

MARINETTE

MAY, J. V.....First National Bank Bldg., OALR

MARSHFIELD

COPPS, LYMAN A..... OALR

MILWAUKEE

BEEBE, C. S.....Wells Bldg., OALR
BLACK, N. M.....Wells Bldg., Op.
CHAPMAN, V. A.....Colby-Abbot Bldg., OALR
GORDON, J. S.....Goldsmith Bldg., OALR
GROVE, W. E.....Wells Bldg., OALR
HEEB, H. J.....Loan and Trust Bldg., OALR
HIGGINS, S. G.....Wells Bldg., OALR
HITZ, H. B.....Goldsmith Bldg., ALR
HOGUE, G. I.....410 Jefferson St., OALR
KRENTZER, A. G.....Trust National Bank Bldg., ALR
MULSOW, J. E.....410 Jefferson St., OALR
RICE, R. H.....Coshell Bldg., Op.AL
SEAMAN, GILBERT E.....309 Goldsmith Bldg., OALR

OSHKOSH

ALLEN, L. P.....19 Jefferson Ave., OALR

RACINE

BRECKENRIDGE, HARRY E.....209 Sixth St., OALR
HOLMES, B. H.....432 Baker Blk., OALR

SUPERIOR

HOVDE, A. G.....Board of Trade Bldg., OALR
MYERS, ELMER A.....Board of Trade Bldg., ALR
SHASTID, T. H.....1920 Johns Ave., OALR
SMITH, R. C.....Board of Trade Bldg., OALR

WAUSAU

SCHLEGEL, HERMAN T.....501 Third St., OALR
WILLARD, L. M.....520 Third St., OALR

WYOMING

CHEYENNE

BECK, F. L.....408 Hynds Bldg., OALR
STRADER, G. L.....408 Hynds Bldg., OALR

AFRICA

NATAL

DURBAN

JOHNSON, LINDSAY.....Britannia Bldg.

CANADA

ALBERTA

EDMONTON

BROWN, W. H.....Bk. of Montreal Bldg., OALR

BRITISH COLUMBIA

VANCOUVER

BOUCHER, R. B.....Banks Bldg., OALR

VICTORIA

KEYS, MORTON J.....307 Jones Block, OALR

MANITOBA

BRANDON

MCDIARMID, H. O.....Rossner Ave. and 9th St., OALR

NOVA SCOTIA

HALIFAX

CUNNINGHAM, ALLAN R.....260 Barrington St., OALR

CUBA

HAVANA

FERNANDEZ, FRANCISCO M.....Prado, 105, OALR

ONTARIO

GUELPH

BENETTO, FREDERICK R.....172 Woolrich St., OALR

HAMILTON

ARRELL, WILLIAM.....317 Main St., E., OALR
MACFARLANE, P. B.....152 James St., S., OALR

TORONTO

DAVIES, T. ALEX.....578 Sherbourne St., OALR
GOLDSMITH, PERRY G.....84 Carlton St., ALR
HILL, C. E.....160 Bloor St., W., Op.
MCCULLOUGH, R. J. P.....102 College St., OALR
MACFARLANE, MURRAY.....190 Bloor St., OALR
MCKELVEY, A. D. M.....160 Bloor St., ALR
RISDON, ERNEST F.....30 Avenue Rd., ALR

QUEBEC

MONTREAL

BIRKETT, H. S.....252 Mountain St., ALR
BALLON, DAVID H.....255 Bishop St., ALR
BOUSQUET, J. P. E.....26 Sherbrook St., W., OALR
LASSALLE, ALBERT.....493 St. Denis St., OALR
MCKEE, S. HANFORD.....158 Crescent St., Op.
ROGERS, J. T.....758 Sherbrook St., W., ALR
ROSENBAUM, J.....206 Bishop St., Op.
WHITE, E. H.....589 Dorchester St., ALR

HAVANA

FERNANDEZ, FRANCISCO M.....Prado, 105, OALR

CZECHO-SLOVAKIA

PRAGUE

ELSCHNIG, ANTON.....Ferdinandstrasse, 10

ENGLAND

LONDON

ELLIOT, LT.-COL. R. H.,
54 Welbeck St., Cavendish Sq., W.
DUNDAS-GRANT.....148 Harley St., W.
LAWFORD, J. B.....99 Harley St., Cavendish Sq., W.
LISTER, SIR WILLIAM T.....24 Devonshire Pl., W. 1.

HOLLAND

LEIDEN

VAN DER HOEVE, PROF. J.....Rijnsburgerweg 6A

SCOTLAND

GLASGOW

GRAY, A. A.....

SWITZERLAND

ZÜRICH

BARKAN, A.....Credit Swisse, OALR

Index photographed at the
beginning for the convenience
of the microfilm user.